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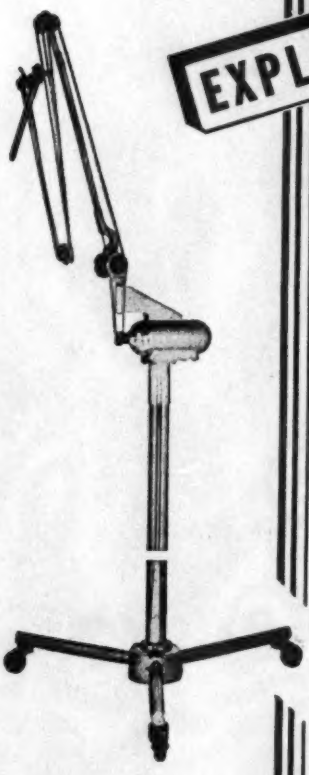

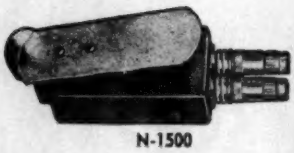

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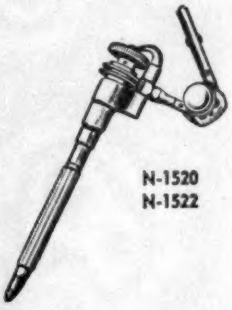

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TABLE OF CONTENTS

VOLUME 64 AUGUST 1956 NUMBER 2

ORIGINAL ARTICLES

PAGE

Mobilization of the Stapes for Otosclerosis
Mervin C. Myerson, M.D., Beverly Hills, Calif. 85

The Temporalis Muscle in Facial Paralysis
William C. Conroy, M.D., Montclair, N. J. 91

Experiences with Peroral Endoscopy in a Growing Midwestern Community
John B. Gregg Jr., Sioux Falls, S. D. 98

Osteomyelitis of the Frontal Bone
C. Stewart Nash, M.D., Rochester, N. Y. 108

The Diagnosis and Treatment of Ménière's Disease
Theo E. Walsh, M.D., St. Louis 118

Objective Audible Clicking in Ear
E. Martin Freund, Albany, N. Y. 129

Cerebrovascular Disease in Otolaryngology
Benjamin H. Shuster, M.D., Philadelphia 134

The Wire Fixation of Autogenous Bone Grafts in the Difficult Rhinoplasty
J. Brydon Smith, M.D., F.R.C.S. (C), Toronto, Canada 140

Osteoma of the Tongue
Ralph Peimer, M.D.; David H. Dreizin, M.D., and Yozo Masugi, M.D., New York 148

AUDIOLOGY SECTION

The Use of Hearing Aids for Severely Deaf Children
T. J. Watson, Ph.D., Manchester, England 151

CASE REPORTS

Hysterical Dysphagia Without Anemia
Adney K. Sutphin, M.D., and Porter P. Vinson, M.D., Richmond, Va. .. 157

CLINICAL NOTES, NEW INSTRUMENTS AND TECHNIQUES

Trial Production of a New Spatula-like Raspatorium for Ligating Larger Blood Vessels
Ryuichi Kubo, M.D., Fukuoka, Japan 159

REGULAR DEPARTMENTS

Abstracts from Current Literature 161
News and Comment 167
Directory of Otolaryngological Societies 169

A. M. A.
ARCHIVES OF OTOLARYNGOLOGY

VOLUME 64 AUGUST 1956 NUMBER 2

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Mobilization of the Stapes for Otosclerosis

A New and Safe Technique

MERVIN C. MYERSON, M.D., Beverly Hills, Calif.

A significant advance in the surgical treatment of otosclerosis was introduced by Rosen [1] in 1953.

Although stapes mobilization had been tried many years before, it remained for Rosen to apply modern methods to this procedure. It had been known since the dissections of Toynbee,[2] that fixation of the stapes at its oval window occurred in a great majority of cases. It remained for Politzer,[3] however, to demonstrate by means of histologic studies of a number of cases of deafness, after death, that otosclerosis was due to a primary disease of the labyrinth.

In 1952, while testing for mobility of the stapes footplate preliminary to the performance of a fenestration operation, Rosen was astonished to find that he had mobilized the stapes and restored the patient's hearing. This experiment caused him to pursue the possibility of mobilizing the fixed footplate as a procedure of choice in suitable cases of otosclerosis. Today, the value of this procedure is well established.* Otologists everywhere are performing the mobilization operation; some are striving to improve the technique and safety of the procedure.

The purpose of this paper is to introduce a technique for stapes mobilization which stresses the factor of safety and aims to simplify the operation. Of great importance is much preparatory work on the fresh cadaver. In addition, a careful perusal of the small volume on the "Mechanism of the Ossicles of the Ear" by Helmholtz [9] will be of great value. In it, among other things, Helmholtz mentions that excursions of the stapes at its foot plate are from 1/18 and 1/14 mm. This emphasizes the very small field involved in mobilization of the stapes. It is obvious that a great deal of force is not necessary to mobilize this structure (Fig. 1).

My routine and technique follow. In order to prevent middle ear infection after operation, strict asepsis is important. Attention is paid to the condition of the nose, nasopharynx, and external auditory canal before surgery. The operation should not be performed in the presence of sinus suppuration or active nasal or nasopharyngeal infection. Regardless of the normal appearance of the upper respiratory passages, the nasal passages are irrigated daily, for three days, before operation with warm saline solution. This is followed by the intranasal administration of tetracycline hydrochloride in nebulized form. The patient is instructed to introduce 3 minims (0.2 cc.) of a 0.125% phenolized oil into each nasal chamber three times a day for one week before surgery. In addition, he is instructed to introduce a dropperful of aqueous solution of benzalkonium (Zephiran), 1:1000, into the external canal three times a day for three days before the operation. For several hours before operation, the ear canal is kept filled with the

Accepted for publication April 24, 1956.

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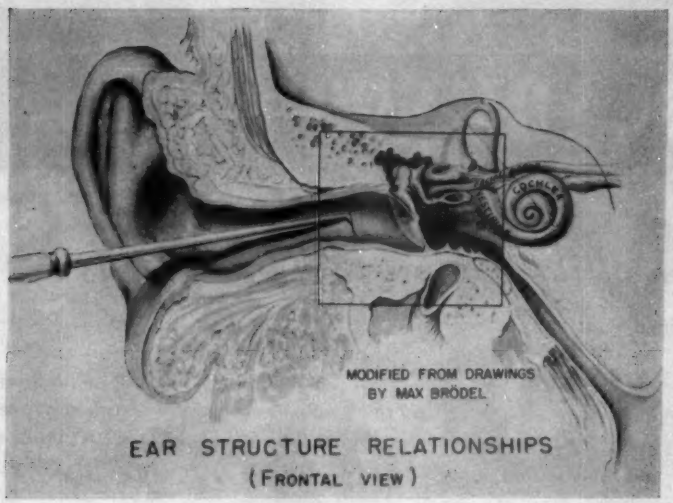

Fig. 1.—Anatomic drawing to show structures involved and their relationships.

same solution. Wetting the skin of the canal facilitates incision into and elevation of the skin and periosteum.

Local anesthesia is produced by the injection of an average of 2 cc. of procaine, 1% solution, each ounce (30 cc.) of which contains 12 minims (0.75 cc.) of epinephrine hydrochloride solution, 1:1000. This concentration of epinephrine hydrochloride has been found ample and satisfactory. The injection is made posterosuperiorly into the retroauricular fold at the junction of the cartilaginous and osseous canals. A single injection will suffice, but additional injection at a point corresponding to the midline of the roof and floor of the canal may be added, if the surgeon does not wish to wait for the solution to infiltrate widely.

The incision is made with two knives; one cuts forward and backward along the midline of the roof and floor of the ear canal, while the other joins these incisions by cutting from side to side. The juncture of these incisions is rounded, not angular (Fig. 2). The incisions reach a point just inside the chondroosseous juncture.

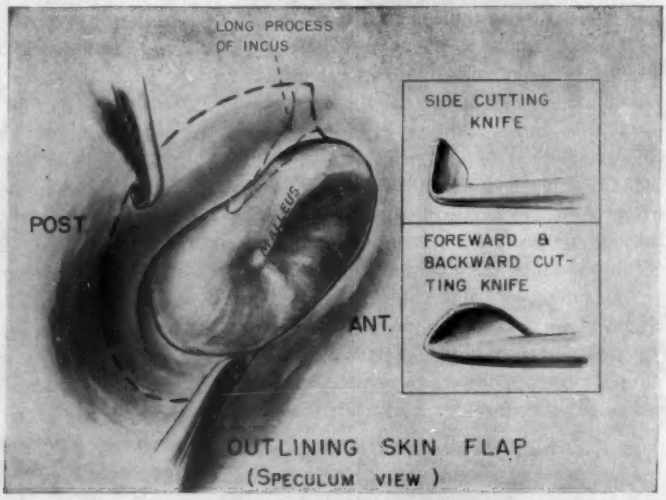

Fig. 2.—Outline of flap, demonstrating use of forward and side-cutting knives.

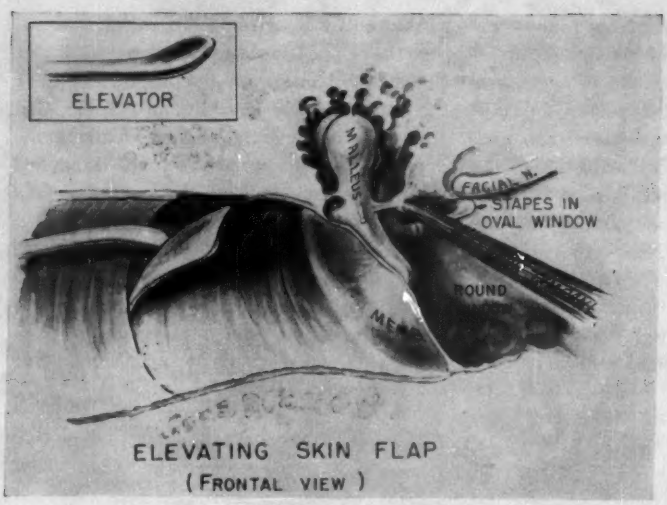

Fig. 3.—Elevation of flap.

Elevation of the flap as outlined is done with two elevators, one curved slightly more than the other. The flatter elevator is better for beginning the elevation. It is usually started posteriorly (Fig. 3). This procedure is facilitated by placing small flat, elongated pieces of cotton, moistened with 2 minims (0.12 cc.) of saline or epinephrine hydrochloride solution between the flap and the bony wall. Gentle pressure is made between the flap and the bony wall, using the dissector or suction tube, as indicated. This prevents tearing of the flap. Elevation is continued until the point of attachment of the drum is reached. At this point the more curved of the elevators is insinuated into the sulcus to which the fibrocartilaginous rim of the tympanic membrane is attached. The membrane is cautiously lifted; the remainder of the rim is readily delivered, and the posterior half of the membrane reflected anteriorly so that the corresponding portion of the tympanic cavity is exposed (Fig. 4). In a majority of temporal bones, the window so outlined permits a good view of the incudostapedial

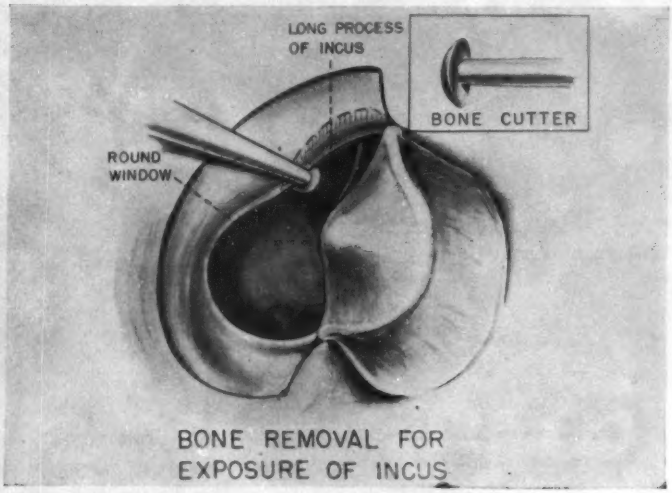

Fig. 4.—Removal of bone with special bone cutter.

articulations. Rosen[6] estimates this to be so in 85% of cases. In the remainder, however, it will be found necessary to remove some of the rim of the posterosuperior canal wall in order to secure such exposure. A rivet-like instrument, similar to the Yankauer Eustachian tube curet, is used for this purpose. Removal of bone should be confined to the region above the point of exit of the chorda tympani nerve from the facial canal, in order to avoid injuring the facial nerve (Fig. 5). A curved pointed instrument, the same as a dental searcher, such as is used by Rosen and others for mobilizing the stapes, is employed to push the chorda tympani nerve up or down, out of the way, and for breaking up visible, fine adhesions if they are present. During the operation suction with fine tubes is applied when necessary to keep the operative field clean.

Once the long process of the incus and the head of the stapes are visualized, a forked instrument (Fig. 6) is fitted over their articulation. The fork is held in position while a specially designed revolving

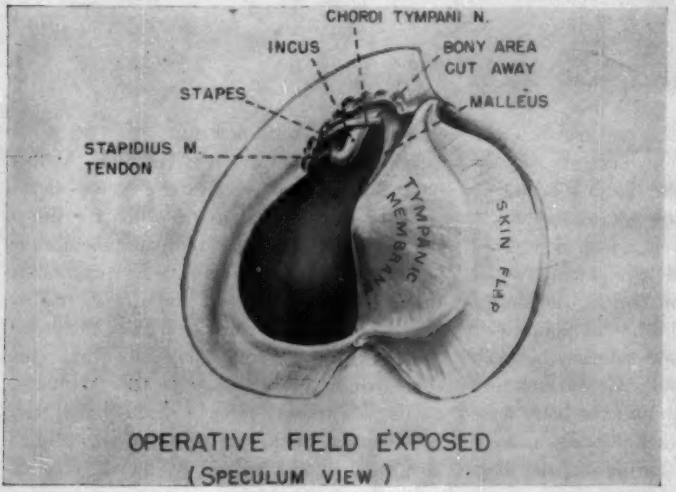

Fig. 5.—Exposure after posterior half of membrane is reflected over.

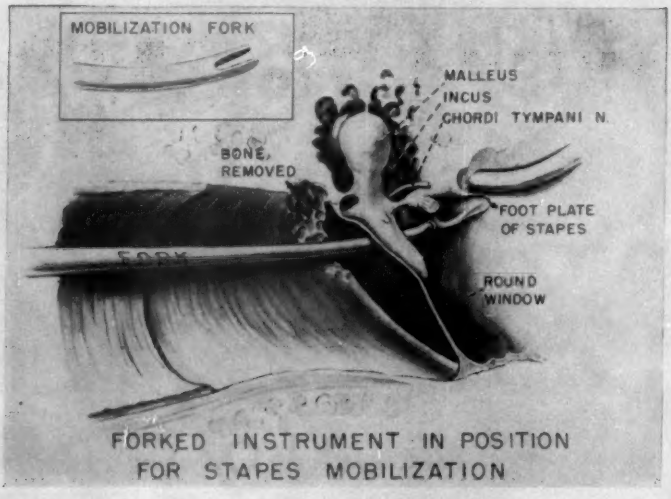

Fig. 6.—Fork placed at incudostapedial articulation.

rod, inserted into a dental handpiece, is brought into contact with it. The purpose of this rod is to create vibrations along the shaft of the forked instrument which are transmitted through the incudostapedial articulation and the crura, to the fixed footplate.

The revolving rod is 3 in. in length. The diameter of the rod is 0.092 in. The flattened portion is in the form of an isosceles triangle, 1¼ in. from base to apex, with the base at the distal end. The flattening reduces the diameter by 0.004 in. The vibrations are delicate or coarse, and generate force and amplitude according to the part of the revolving rod in contact with it. The force and amplitude depend upon the number of revolutions per minute of the dental handpiece and the inserted rod, and the part of the rod which is applied against the shaft of the forked instrument by the operator. No perceptible pressure should be applied against the shaft of the forked instrument with the rod. One should rely entirely upon the three speeds supplied by the foot switch. The low and medium speeds should be tried first. If they do not prove successful, the high speed should be used. At low speed approximately 1500 rpm are delivered; with medium speed approximately 5000, and with high speed † approximately 9000 rpm are produced. It would seem from work on the cadaver and from clinical experience that 9000 rpm will mobilize any stapes that can be dislodged, short of causing irreparable damage (Fig. 7). It would appear that, despite firm fixation of the footplate, the crura will not break if this technique is used. This is because the vibrations are transmitted through the temporal bone and the skull beyond the region of the oval window.

Several stapes were sunk into plaster of paris at the footplate. The plaster mass was then placed upon a cushion of foam rubber on a table simulating the operating table. The ossicle was subjected to vibrations transmitted through the forked vibrator by subjecting its shaft to contact with the rotating rod at 9000 rpm. The crura of the stapes did not break, proving that the vibrations do not stop at the footplate, but go beyond the structures immediately involved. Of interest is the fact that fine linear fractures of the plaster were seen at the point of insertion of the footplates, with magnifying lenses.

In a certain number of otosclerotic middle ears there may be adhesions between the incus and malleus or the incus and stapes, or both. The vibrations transmitted through

† At present 9000 rpm are being used routinely.

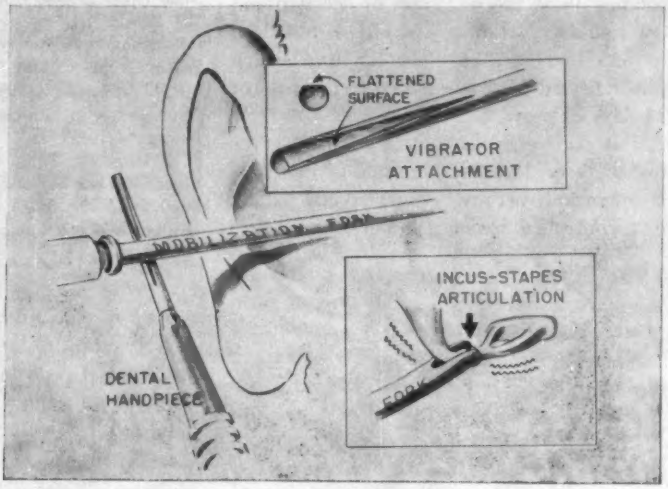

Fig. 7.—Revolving rod in dental handpiece applied to shaft of forked instrument.

the forked rod have an added purpose: to break up these adhesions should the fixation not be permanent.

If a result is not obtained after use of the vibrating technique described, the direct method of manipulating the footplate according to Rosen should be tried. If this fails, one should resort to the fenestration operation of Lempert.

An arbitrary limit should be established regarding the amount of force which should be applied to the fixed stapes footplate. At the present time, vibrations transmitted to the footplate which are generated by a specially flattened rod revolving 9000 times per minute would seem to suit this requirement. Mobilization of the stapes should be performed with the realization that many footplates cannot be mobilized within the limits of safety, and with the assurance of improved hearing.

When the incudostapedial manipulation is completed, the flap is pulled back and pressed gently into position. A piece of nitrofurazone (Furacin) gauze is placed over the line of incision as far inward as the tympanic membrane. The gauze is removed the next day. No further dressing is necessary. A small piece of cotton is placed in the external meatus to prevent the patient from touching the inside of the canal. Healing is completed within two to five days, depending upon the condition of the flap and the amount of trauma to which it has been subjected. That part of the ear drum which was reflected does not return to normal for 14 days or more.

The patient is kept in the hospital for two or three days. A minimum of two days is important, because a hospitalized patient is a controlled patient, and it is necessary to control him for 48 hours to guarantee as much as possible against infection. The patient is warned not to blow his nose until he is told that he may do so. He is also admonished to keep his finger or any other object away from his ear canal. For this reason he is advised to keep a small piece of absorbent cotton loosely in the meatus.

Summary

A procedure is outlined which affords a greater degree of safety than has heretofore been available for mobilization of a fixed stapes footplate in otosclerosis.

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REFERENCES

1. Rosen, S.: Mobilization of the Stapes to Restore Hearing in Otosclerosis, New York J. Med. 53:2650, 1953.
2. Toynbee, J.: Pathological and Surgical Observations on Diseases of the Ear, Med.-Chir. Tr. 24: 190, 1841.
3. Politzer, A.: Über primäre Erkrankung der knöchernen Labrynthskapsel, Ztschr. Ohrenh. 25: 309, 1893-1894.
4. Rosen, S., and Bergman, M.: Mobilization of the Stapes for Otosclerotic Deafness, A.M.A. Arch. Otolaryng. 61:197, 1955.
5. Rosen, S., and Bergman, M.: Restoration of Hearing in Otosclerosis by Mobilization of the Fixed Stapedial Footplate, Laryngoscope 65:224, 1955.
6. Rosen, S.: Mobilization of Stapes, J. Mt. Sinai Hosp. 23:200, 1956.
7. Scheer, A. A.: Restoration of Hearing in Otosclerosis by Transtympanic Mobilization of the Stapes, A. M. A. Arch. Otolaryng. 61:513, 1955.
8. Meurman, Y., and Meurman, O.: Stapes Mobilization in Otosclerosis, A. M. A. Arch. Otolaryng. 62:164, 1955.
9. Helmholtz, H. L. F.: The Mechanism of the Ossicles of the Ear and Membrana Tympani, translated by A. H. Buck and N. Smith, New York, William Wood & Company, 1873.

The Temporalis Muscle in Facial Paralysis

Report of a Case

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Introduction

In the surgical treatment of facial paralysis, support of facial musculature by static or dynamic means must be relegated to the procedure of last resort, since the mimetic character of facial musculature cannot be restored except by reestablishment of the integrity of the facial nerve by direct anastomosis or nerve graft. Anastomosis of peripheral branches of an interrupted facial nerve, when effected early, to the proximal portion of a lower cranial nerve restores normal tone to the previously paralyzed facial muscles, and, although emotional response is lacking and "mass action" often produces grotesque facial grimaces, the facial symmetry is, at least, fairly normal in repose. However, when the aforementioned measures have been unsuccessful or are contraindicated by long-standing paralysis with atrophy and fibrosis of the facial musculature, to the plastic surgeon falls the task of rehabilitating a person who presents, on one side, to the world a sagging, lifeless mask. Results achieved by the replacement of at least 10 delicate muscles normally possessing mimetic in addition to voluntary activity by the crude techniques at our disposal must be interpreted with understanding of the monumental task involved. The dissatisfaction with present techniques is evidenced by the great variety of methods employed in the correction of inveterate facial paralysis, recently reviewed by Harris.[1] It is my purpose to present a procedure, previously undescribed, which I feel is logical in approach, simple in performance, and provides the patient with a desirable dynamic crutch for his facial deformity without increasing the present or adding secondary deformity.

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Review

In peripheral facial paralysis, the forehead on the affected side is flat without normal creases, the eyebrow is low, the palpebral fissure is wider than the unaffected side, the lower half of the nose deviates slightly to the normal side, the affected angle of the mouth droops, and, most prominently, the nasolabial groove is absent. Aside from the upper lid, the whole is devoid of motion and, in talking or emotional demonstration, the whole lower half of the face shifts markedly to the normal side. Subjectively there is epiphora and easy fatigue of the affected eye. Although possessing normal sensation, because of immobility, the half of the face feels "dead." During mastication, food collects uncomfortably against the flaccid buccinator.

Attempts to animate the forehead and eyelids by muscle or muscle-fascia transplants have resulted in exaggeration of a previously slight deformity in this region, and, although slight movement resulted, it was neither natural nor protective nor effectively supported the lower lid. A lateral tarsorrhaphy of either edge-to-edge or overlapping variety combined with the removal of an ellipse of skin including frontalis muscle to the periosteum from the forehead corresponding to the most prominent contralateral crease narrowed the palpebral fissure, raised the eyebrow, and ameliorated the flatness of the forehead, resulting in a gratifying improvement in appearance of the upper half of the face.

The major deformity is found in the

lower half of the face. It has been demonstrated[2] that, in the face, the prominent creases exist at right angles to the longitudinal direction of the muscle fibers. The nasolabial groove may be construed as the facial crease at right angles to the elevator muscles of the lips, and a line at a right angle to the center of this line gives us an approximate resultant line of force exerted by the contraction of these muscles. Any single force elevating the lips must act along this line if a natural static or dynamic elevation of the affected lips is to be achieved. Another important factor is that the buccinator, innervated by the facial nerve, acts to pull the lateral lips backward and slightly upward against the premolar and molar teeth and modifies the aforementioned line of force, adding a posterior to the upward and outward direction of "pull."

Those procedures utilizing fascia lata extending from the lips to the temporalis fascia or zygoma take advantage of only the former of the above considerations. The masseter transplants are objectionable because the line of force approaches the horizontal and has no posterior component and dissection of the muscle segment to the level of origin to provide a higher line of pull jeopardizes the contractibility of the transplant. In my experience with this procedure, the affected half of the lips droops in repose and, on contraction, although the affected oral commissure is pulled laterally, the original deformity is increased by an unnatural convexity of the affected cheek.

The temporalis muscle with the full extent of its tendon acting under the zygomatic arch provides, in contraction, a force which most closely approximates that caused by the sum of the labial elevator and buccinator muscles. McLaughlin,[3] utilizing the temporalis muscle and included sectioned coronoid process, has achieved a very satisfactory cosmetic result on voluntary contraction of the temporalis muscle. Certain anatomical and physiological considerations have caused me to modify this technique.

Batson,[4] in a dissertation on the temporalis muscle, has pointed out that the anterior portion of this bipinnate muscle contains fibers 30-31 mm. in length contracting superiorly to a maximum extent of 1.5 cm., whereas the posterior portion possesses fibers of half that length and range of contraction with a force directed posteriorly. Also of importance is that the region of the coronoid process from about a centimeter above the zygomatic arch to the greater portion of the insertion of the temporalis tendon consists of an investment of loose areolar connective tissue, so that the coronoid process moves in this potential space much like a piston in a cylinder. This "space" is easily approached surgically through a fascial incision immediately above the zygomatic arch.

A muscle belly acting at a distance, as found in the extremities, is most efficient acting via a tendon circumferentially complete and encased in paratenon. Although the iliotibial band, from which fascial strips used in repair of facial paralysis are obtained, is a flat tendon, its strips are not circumferentially encased in paratenon and adhere to surrounding structures when transplanted, if not replaced by fibrous tissue. The tendons of the extensor digitorum longus of the toes are fine, cord-like structures, circumferentially complete, easily and quickly obtained without the use of special instruments, and incur no donor deformity beyond a vertical scar on the dorsum of the foot and a small transverse scar above the ankle.

Report of a Case

A 26-year-old sailor on March 22, 1953, sustained, in an automobile accident, a depressed skull fracture of the left temporal region, resulting in a cranial defect and left peripheral facial paralysis. A tantalum plate was placed in the cranial defect in December, 1953. In February, 1954, after repeated neurosurgical and physical-medicine evaluations, it was concluded that complete paralysis of the facial nerve was present without evidence of regeneration and surgical support was indicated.

Following shaving of the face and the left temporal region and the administration of preoperative

Fig. 1.—Preoperative views of the patient.

Fig. 2.—Procuring toe tendons.

Fig. 3.—Tendons to second, third, and fourth toes with common tendon belly.

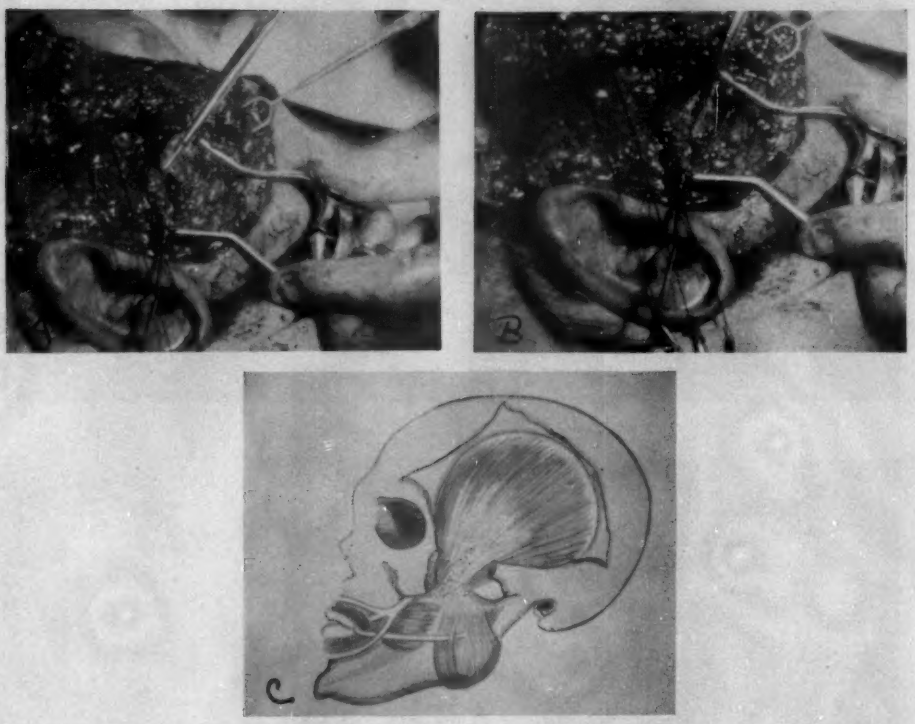

Fig. 4.—"Face-lift" skin flap retracted forward. Clamp on anterior half of temporalis tendon. *A*, relaxed muscle; *B*, contracted muscle. *C*, diagram showing course of tendons attached to anterior half of temporalis tendon.

sedation, on March 1, 1954, a spinal anesthetic was given and the face, left leg, and foot were prepared and draped. A 7 cm. longitudinal incision was made down to the deep fascia on the dorsum of the left foot. The extensor digitorum longus tendons to the second, third, and fourth digits were exposed, sectioned at the metatarsophalangeal joint, and raised by sharp dissection with most of their paratenon up to the cruciate ligament. Above the ankle a transverse incision 1.5 cm. in length was made over the common tendon, and the dissected tendons were attached to a probe and passed proximad under the cruciate ligament and out of this wound and severed from the main tendon belly. Both wounds were then closed in layers.

The left side of the face from 1 in. below the body of the mandible to the temporal region, including both lips across the midline and deeply to the coronoid process region, was infiltrated with 1% procaine with 8 drops of 1:1000 epinephrine per ounce. A modified "face-lift" incision was made beginning within the hairline 1 in. anteriorly and slightly above the superior auricular attachment, progressing posteriorly to that attachment, inferiorly along the preauricular crease, hooking around the ear lobe, and then continuing anteriorly parallel to the cervical creases for about 2 in. A facial flap was raised, exposing the subcutaneous tissues to within 1.5 cm. of the left oral commissure and the malar eminence, and made just lateral to the orbit, exposing the temporalis fascia or aponeurosis. Following satisfactory hemostasis an incision was made transversely in the temporalis fascia immediately above the zygomatic arch over the coronoid process. Retraction exposed a "space" filled with loose areolar connective tissue and the tip of the coronoid process. The patient was directed to bite down, and a stout, firm tendon belly attached to the coronoid process appeared. Slightly less than the anterior half of the tendon was bluntly dissected free for about 1 in., with respect for underlying vessels, and then sectioned at the tip of the coronoid process. At this point the free end of the tendon was grasped in a clamp, and voluntary excursion of the tendon end of about 1.5 cm. was observed. Loose areolar tissue from the medial side of the tendon was then sutured across the raw area of the separated ten-

Fig. 5.—Immediate postoperative views: *A*, note fine nonabsorbable surgical sutures in lips and "bolsters" of lateral tarsorrhaphy. *B*, closure of "face-lift" incision.

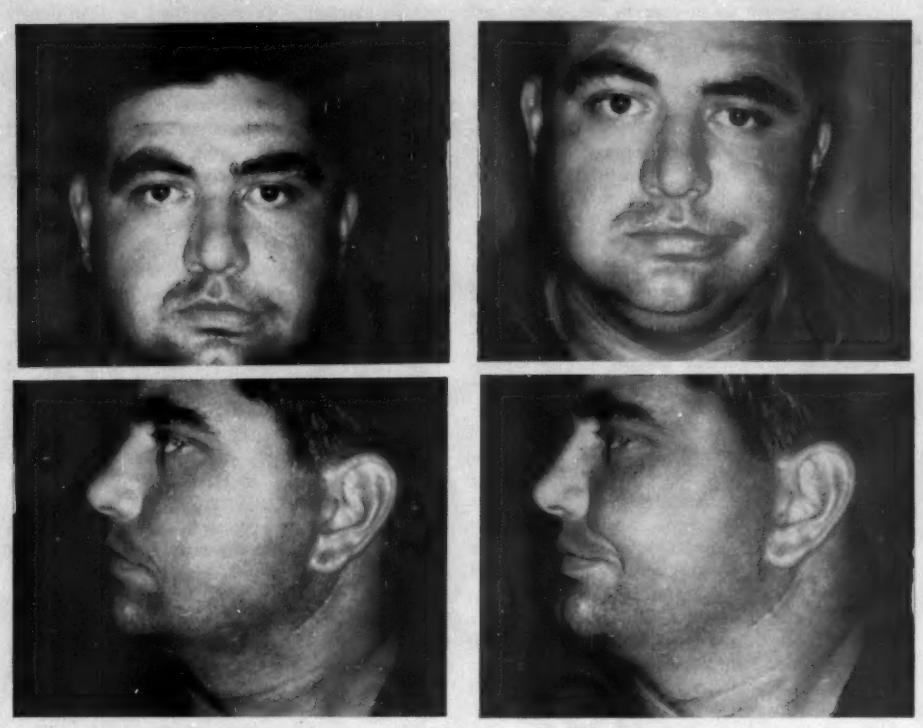

Fig. 6.—Views taken five months postoperatively: Upper left, temporalis relaxed. Upper right, temporalis contracted. Lower left, temporalis relaxed. Lower right, temporalis contracted.

don and anchored to the temporalis fascia with 0000 nonabsorbable surgical (silk) sutures.

A blunt probe was then passed under the zygomatic arch from the fascial opening and made to emerge at the edge of the previous undermining at the angle of the mouth. A double strand of 000 nonabsorbable surgical suture was attached to the probe and threaded through this tunnel, and clamps were placed on either end. One centimeter vertical skin incisions were then made in the upper and lower lips just to the normal side of the midline. The blunt probe was passed through the in-

cisions and laterally through the orbicularis muscle to emerge at the undermined area at the angle of the mouth in the region of the lower end of the suture passed under the zygomatic arch. The tunnels created were similarly threaded with 000 nonabsorbable surgical suture. A third vertical incision was made in the nasolabial fold skin lateral to the angle of the mouth on the paralyzed side. The blunt probe was passed from the undermined area around the orbicularis muscle from behind to emerge at the incision and was then passed from the incision to the undermined area, and this tunnel looping around the orbicularis muscle was similarly threaded with 000 nonabsorbable surgical suture. The nonabsorbable surgical sutures extending into the undermined area from the labial tunnels were then tied to the ends of the tendon slips previously procured and pulled through the tunnels. The tendon slip passed from behind the orbicularis muscle to emerge in the undermined area was implanted in the masseter muscle just below the zygoma and sutured there with 0000 nonabsorbable surgical suture. The common tendon was then attached to the inferior end of the suture passed under the zygomatic arch and then pulled up under that structure to emerge at the opening in the temporalis fascia. This tendon was then passed through an opening or button hole bluntly created in the partially freed anterior half of the temporalis tendon. Traction was then made on the toe tendon so that the corner of the mouth was well overcorrected, and firm countertraction was placed on the temporalis tendon. The two tendons were interlaced with a 0000 nonabsorbable surgical suture, the excess toe tendon excised, and the severed end buried. The upper and lower lips were then advanced superiorly and laterally to moderate overcorrection on the protruding tendons. After all tendons to the orbicularis oris muscle were anchored with 0000 chromic absorbable surgical (gut) suture, the excess tendons at the center of the lips were excised and all three wounds closed in layers with interrupted 0000 absorbable surgical suture subcutaneously and 000000 nonabsorbable surgical suture to the skin. The "face-lift" incision was then closed with subcutaneous sutures of 0000 chromic absorbable surgical suture and interrupted 00000 nonabsorbable surgical suture to the skin after the excision of excess. A Penrose drain was placed in the lower angle of the wound. Prior to complete wound closure, satisfactory tendon position was checked by having the patient contract the transplant. Finally the lateral 0.5 cm. of the palpebral borders of the affected lids were denuded of epithelium and fixed to each other with 0000 nonabsorbable surgical suture over bolsters. Following the application of Ivy loops to the anterior incisors and an intermaxillary wire to provide immobility of the jaws, moderate pressure dressings were applied to the operative sites. At placement of the loops it was noted that the lower-lip tendon had entered the oral cavity in 1 cm. of its course. This was not observed at the time owing to intentional care to prevent contamination of the operative field with oral fluids. The tendon was repositioned and all wounds closed. A Levin tube was placed for nutritional purposes.

On return to the postoperative ward 1,000,000 units of penicillin was given, followed by 300,000 units daily for six days. All facial activity was prohibited for one week. All wounds healed uneventfully. The Penrose drain was removed on the first postoperative day, and the Levin tube and all sutures were removed on the sixth postoperative day. The wound integrity was protected with collodion strips for two weeks. After three weeks the intermaxillary fixation was removed, masticatory activity was encouraged, and daily faradic stimulation of the anterior half of the temporalis muscle together with reeducation of that muscle was instituted. Serial weekly photographs were taken.

On April 5, 1954, with the patient in a sitting position, the left eyebrow was raised to the level of the normal side and a transverse ellipse marked out on the forehead corresponding to the most prominent contralateral crease. After local preparation and procaine hydrochloride (Novocain) infiltration, the outlined area 4 cm. long and 1.3 cm. wide was excised down to the periosteum and the wound closed with interrupted subcutaneous 0000 chromic absorbable surgical sutures and interrupted 000000 nonabsorbable surgical sutures to the skin.

Penicillin, 300,000 units, was given intramuscularly for three days postoperatively. On the fourth postoperative day the skin sutures were removed and collodion strips applied.

Five months postoperatively a satisfactory reanimation of the lower half of the paralyzed face had been obtained. The elevation of the left angle of the mouth on voluntary contraction approximated 1.5 cm. two months postoperatively and improved but slightly thereafter. However, serial photographs revealed a progressive improvement in appearance and balance of the face at rest. Supervised daily reeducation of the temporalis transplant has produced a fair voluntary balanced smile as far as the lower half of the face is concerned, provided the teeth are in occlusion.

In talking and ungoverned emotional expression, the preoperative distortion has been greatly reduced, but, as expected, lack of participation of the affected face is evident. No ability to disassociate the transplanted muscle has been noted.

Subjectively, the patient was very pleased with the improvement in facial appearance and the control, although limited, of the paralyzed face. Eye fatigue had been much reduced, and epiphora occurred only on exposure to winds. The affected face, through ability to move, no longer felt dead.

On mastication food no longer collected against the flaccid buccinator.

A recent communication from the patient, two years after surgery, notes that the face "looks good and doesn't sag."

Summary

The treatment of inveterate facial paralysis by surgical support is briefly reviewed, setting forth anatomical considerations which render the anterior half of the temporalis muscle, by configuration and position, the motor unit of choice in animating the lower half of the paralyzed face. A case of facial paralysis is presented and discussed in which a satisfactory result was obtained by utilizing the anterior half of the temporalis tendon in combination with the extensor digitorum longus tendons to the toes. The upper half of the face was improved by a lateral tarsorrhaphy and the excision of forehead skin. Preoperative, operative, and postoperative photographs are included.

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REFERENCES

1. Harris, H. I.: Plast. & Reconstruct. Surg. 13:193, 1954.
2. Rubin, L. R.: Plast. & Reconstruct. Surg. 3:147, 1948.
3. McLaughlin, C. R.: Plast. & Reconstruct. Surg. 11:302, 1953.
4. Batson, O. V.: Oral Surg. 6:40, 1953.
5. Fox, S. A.: Ophthalmic Plastic Surgery, New York, Grune & Stratton, Inc., 1952, p. 71.
6. Brown, J. B.: Ann. Surg. 109:1016, 1939.
7. Owens, N.: Plast. & Reconstruct. Surg. 7:345, 1951.
8. Brunner, H.: Plast. & Reconstruct. Surg. 8:390, 1951.
9. Clarkson, P.: Brit. J. Plast. Surg. 5:259, 1953.
10. Adams, W. M.: Plast. & Reconstruct. Surg. 1:216, 1946.
11. Battle, R. J. V.: Brit. J. Plast. Surg. 5:247, 1953.

Experiences with Peroral Endoscopy in a Growing Midwestern Community

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In the past many reports and series of cases have emanated from endoscopy clinics of this country and elsewhere. These studies generally reflect the experiences of groups located in large-population areas. Although the information derived from these studies is extremely valuable for instruction and for statistical data, it does little to let the young endoscopist, entering practice, know what to expect if he goes into a smaller community. Unfortunately, the experience of the endoscopist in a smaller community is rarely documented because he usually dies before he has the chance to compile and publish the findings of his life's work. However, some reports * are available from smaller endoscopy centers. These do much to bridge the gap in experiences from the large "Clinic" to private practice of endoscopy.

Recently death brought to a close the career of one of South Dakota's pioneers in the field of laryngobronchoesophagology, Dr. John B. Gregg, Sioux Falls, S. D. Dr. Gregg received his endoscopic training at the L. W. Dean Clinic in Iowa City, 1915-1920. Because this field of medicine, especially foreign-body endoscopy, had always been one of his major interests and hobbies, he left behind him carefully documented records of his findings. With these data available it would seem prudent to pause briefly and examine the scope and development of this facet of medicine in this growing community (1921: population 25,000; 1954: population 60,000).

In the earlier part of this work, endoscopic procedures were done oftener for foreign bodies in the air and food passages. In recent years an increasing amount of diagnostic work was done, and, as would be expected, the volume of endoscopic work, both foreign body and diagnostic, increased proportionately with the increase in population (Table 1). Yet, even with the increasing number of diagnostic studies in recent years, the bulk of the endoscopies were done because of extraneous material in the air and food passages.

During the 33-year period included in this study, there were 523 endoscopic procedures performed (Table 3). Of these, there were 184 bronchoscopies, during which 103 foreign particles were recovered. There were 250 esophagoscopies, with 204 proved foreign bodies, and 89 laryngopharyngoscopies, of which 61 had foreign material present. Six malignant lung neoplasms were found, ten malignant esophageal neoplasms were diagnosed, and eight malignant laryngopharyngeal neoplasms were recorded. It is of interest that the majority of the malignancies were diagnosed in the past 10 years. Negative examinations were reported in 37 bronchoscopies, 15 esophagoscopies, and in 1 laryngopharyngoscopy. Other pathology, which included bronchial and esophageal stenosis, tracheobronchitis, atelectasis, congenital malformation of the larynx and bronchial tree, cardiospasm of the esophagus, lye stricture of the esophagus, and others, was found in 34 instances. In six cases foreign particles were found in the esophagus of patients with some form of stricture formation. Three patients with esophageal foreign body and three with tracheobronchial for-

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*References 1-4.

TABLE 1.—*Endoscopic Procedures Done Per Five-Year Period* (1920-1954)

Years	Bronchoscopies	Esophagoscopies	Laryngoscopies	Total
1920–1925	9	12	5	26
1926–1930	9	19	3	31
1931–1935	30	34	15	79
1936–1940	14	31	20	65
1941–1945	28	32	11	71
1946–1950	45	66	21	132
1950–1953	49	56	14	119
Total	184	250	89	523

TABLE 2.—*Foreign Bodies: Type, Location, Age Range, and Time Range*

Type	Age Range	Duration or Time Range	Laryngo-pharynx	Bronchus	Esoph-agus	Total
Meat bolus	4 ½–70 yr.	1 hr.–3 days	1	1	19	21
Bone, chicken	15 mo.–85 yr.	3 hr.–6 days	5	1	33	39
Bone, duck	63 yr.	38 hr.			1	1
Bone, fish	2 ½–80 yr.	30 min.–2 ½ mo.	32		10	42
Bone, pheasant	15 mo.–59 yr.	1 hr.–4 days	2		8	10
Bone, pork, beef	11 mo.–73 yr.	6 hr.–2 days		1	18	19
Bone, turkey	50–60 yr.	6 hr.–6 days			2	2
Salmon vertebra	5 yr.	5 days			1	1
Oyster shell	62 yr.	16 hr.			1	1
Tooth, human	12 yr.	4 ½ mo.		1		1
Egg shell	10 mo.	62 hr.	1			1
Penny	7 mo.–3 ½ yr.	6 hr.–4 days			13	13
Nickel	18 mo.–6 yr.	7 hr.–2 days			10	10
Dime	12 yr.	24 hr.			1	1
Quarter	16 mo.–7 yr.	22 hr.–7 days			3	3
Half dollar	9 yr.	18 hr.			1	1
Silver dollar	16 yr.	26 hr.			1	1
Safety pin	4 mo.–3 yr.	2 hr.–15 days	5	2	21	28
Common pin, needle	2 ½–18 yr.	10 hr.–17 hr.	4	1	3	8
Thumbtack	14 mo.–13 yr.	5 hr.–2 days	1	1	5	7
Pen point	11 yr.	2 hr.			1	1
Button	13 mo.–2 yr.	3 hr.–6 wk.			7	7
Metal disc	18 mo.–4 yr.	4 hr.–11 days			10	10
Wire key	2 yr.	1 mo.			1	1
Earring	10 mo.–3 yr.	4 hr.–3 wk.			2	2
Wire	10 mo.–60 yr.	4 hr.–6 wk.	4	1	1	6
Hair pin	2 yr.	48 hr.			1	1
Balloon whistle	10–12 yr.	10–24 hr.			2	2
Nail	3–5 yr.	4–38 hr.	1	2		3
Rivet	3 yr.	12 hr.			1	1
Tin foil	2 ½–8 yr.	4 days–2 wk.		1	1	2
Toy watch	8 yr.	14 hr.			1	1
Jackstone	7 yr.	4 hr.			1	1
Pencil top	5 yr.	15 hr.		1		1
Screw	3 ½ yr.	13 mo.		1		1
Metal tube	11 mo.	24 hr.		1		1
Metal toy	10 yr.	1 hr.	1			1
Plastic cylinder	6–18 yr.	4–21 hr.		1	1	2
Comb	6 mo.	8 hr.	1			1
Rubber tube	40 yr.	4 mo.		1		1
Glass 3	43 yr.	3 mo.	1			1
Gentian violet pill	32 mo.	2 days		1		1
Porcelain	18 mo.–2 yr.	1 hr.–1 mo.		2		2
Wax crayon	22 mo.	9 days		1		1
Piece of match	29 yr.	3 days		1		1
Toothpick	2 ½ yr.	5 days		1		1
Piece of gravel	17 mo.	5 wk.		1		1
Peanut	1–14 yr.	3 hr.–2 mo.		55	1	56
Corn	15 mo.–4 yr.	13 hr.–27 days	1	10		11
Bean	15 mo.–2 ½ yr.	5–48 hr.		3		3
Popped corn	8 mo.–40 yr.	12 hr.–3 days		4		4
Carrot	3–4 yr.	72 hr.–4 days		3		3
Hair barrette	16 mo.	5 hr.			1	1
Celluloid toy	1 yr.	5 hr.		1		1
Watermelon seed	12 mo.	17 days		1		1
Sand burr	6 mo.–35 yr.	1–12 hr.	1	1		2
Pear pulp	19 yr.	48 hr.		1 (myesthesia gravis)		1
Peanut candy	16 yr.	52 hr.			1	1
Plum, peach pit	15–18 yr.	14 hr.–3 days			3	3
Almond shell	16 yr.	24 hr.		1		1
Vomitus	32 yr.	1 ½ hr.		1		1
Cellophane	11 mo.	6 days		1		1
Escaped into stomach or coughed out			60	106	187	353
				2	18	
		Totals	60	108	205	353

TABLE 3.—*Endoscopic Findings*

Bronchoscopy	
Foreign body	105
Neoplasm, malignant	6
Neoplasm, benign	1
Other pathology	31
Aspiration bronchoscopy	4
Negative	37
Total	184
Esophagoscopy	
Foreign body	204
Neoplasm, malignant	10
Neoplasm, benign	1
Other pathology	20
Negative	15
Total	250
Laryngopharyngoscopy	
Foreign body	61
Neoplasm, malignant	8
Neoplasm, benign	9
Other pathology	10
Negative	1
Total	89
Total endoscopic procedures	523

eign body had to be reexamined before the intruding particle was found.

As might be anticipated, in this group of patients, the majority of the bronchial foreign bodies were recovered from children, the ratio being 14:1, children to adults. Adults surpassed children 3:2 in the frequency of esophageal foreign bodies. In children small toys, coins, pieces of peanuts, and safety pins were the objects which were found oftenest. Small bones and pieces of poorly chewed meat comprised the largest groups of foreign particles recovered from adults. The various foreign particles, their locations, the age range in which they were found, and the range of the length of time they were in place are summarized in Table 2. Some of these foreign bodies are pictured in Figures 1, 2, 3, 4, and 5.

The anatomic location of foreign particles in the bronchial tree was as follows: trachea 16%, both main bronchi 2.5%, right main bronchus 45.5%, left main bronchus 24.5%, right lower lobe bronchus 5%, left lower lobe bronchus 3.7%, right middle lobe bronchus 1.6%, and left upper lobe bronchus 1.2%. In the esophagus alien particles were located in the upper portion in 58%, in the midportion in 13%, and in the lower in 29% of the cases.

The alien particle which was in the bronchial tree for the longest time was a screw, located in the right main bronchus of a 3½-year-old boy for 13 months. The average duration of tracheobronchial foreign bodies in this group of patients was about 12 hours. The esophageal foreign body which was in place the longest was a button, lodged in the upper esophagus of a 2-year-old girl for six weeks. The average duration of foreign material in the esophagus was about 24 hours, and the average duration of particles in the laryngopharynx was about eight hours. In one

Fig. 1.—Coins removed from the hypopharynx and esophagus.

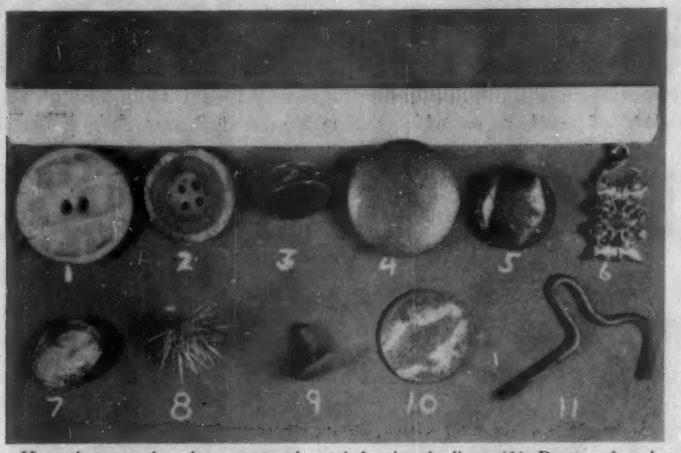

Fig. 2.—Hypopharyngeal and upper esophageal foreign bodies: (1) Button, female, aged 10 years, 4 days' duration. (2) Button, female, 17 months and 6 weeks. (3) Overall button, female, 16 months and 24 hours. (4) Button, female, 4 years and 16 hours. (5) Button, female, 16 months and 3 hours. (6) Pendant, female, 10 years and 1 hour. (7) Plum pit, female, 15 years and 14 hours. (8) Sand burr, male, adult, 12 hours. (9) Rivet, male, 3 years and 5 days. (10) Tax token, male, 2 years and 5 days. (11) Wire key, male, 3 years and 1 month.

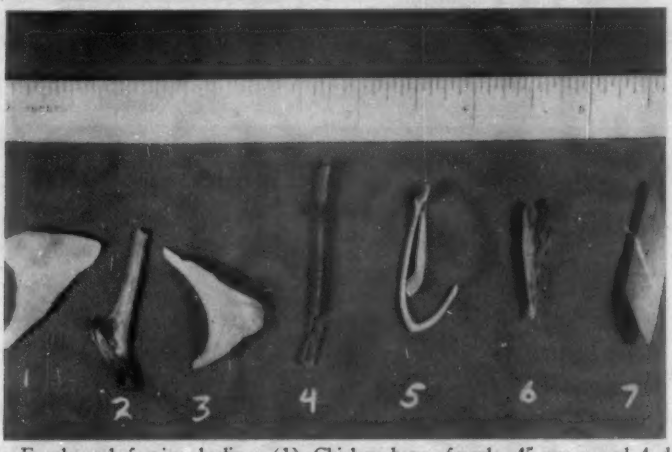

Fig. 3.—Esophageal foreign bodies: (1) Chicken bone, female, 45 years and 4 days. (2) Chicken bone, male, 40 years and 48 hours. (3) Pork bone, female, 40 years and 15 hours. (4) Chicken bone, female, 50 years and 3 days. (5) Squab wish bone, female, 45 years and 36 hours. (6) Pork bone, male, 55 years and 14 hours. (7) Chicken bone, male, 42 years and 2 hours.

case a fishbone was lodged in the lingual tonsil for two and one-half months. There was no special sex preponderance of foreign material in adults or children. The youngest patient with a foreign body was a 4-month-old child with a safety pin in the upper esophagus. The oldest patient was an 85-year-old woman with a chicken bone lodged in the upper esophagus. The largest object ingested was a silver dollar, which was removed from the upper portion of the esophagus of a 16-year-old boy 26 hours after ingestion. The smallest particles were the pieces of peanuts, fragments of fishbones, etc. The most difficult and time-consuming foreign particles were the open

safety pins, situated point up, located in the hypopharynx and esophagus. With the advent of the antibiotics, treatment of this type of ingestant became somewhat easier.

In this series of cases there were six who died. Of these, one was brought to the hospital dead because of obstruction to the airway by a piece of weiner in the larynx. One patient died on the operating table during bronchoscopy; one died during

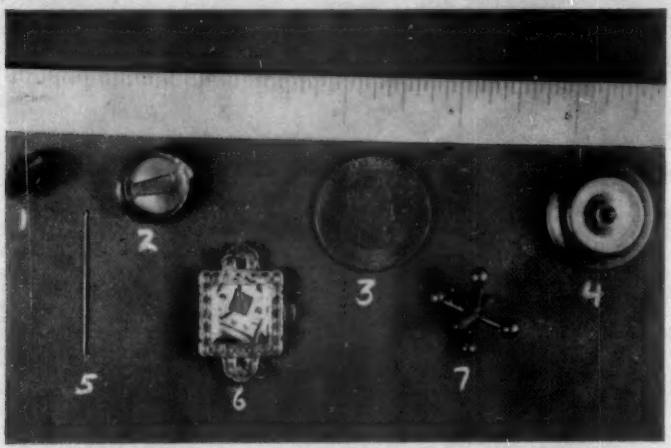

Fig. 4.—Esophageal foreign bodies: (1) Thumbtack, male, 1½ year and 5 hours' duration. (2) Balloon whistle, female, 12 years and 10 hours. (3) Metal medal, male, 2½ years and 48 hours. (4) Toy car wheel, female, 4 years and 24 hours. (5) Needle, female, 5 years and 17 hours. (6) Toy watch, female, 8 years and 14 hours. (7) Jackstone, female, 7 years and 4 hours.

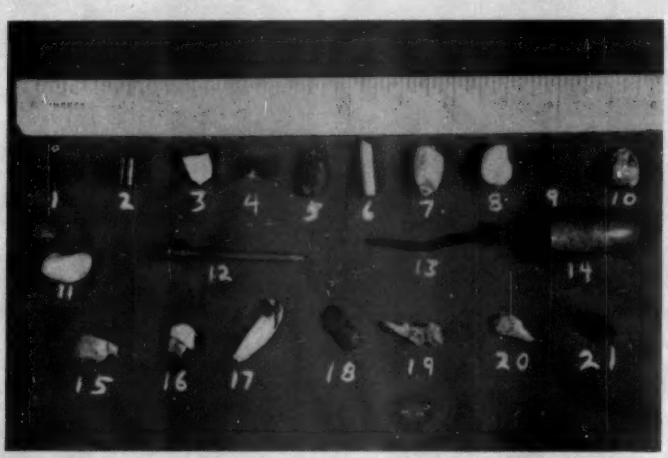

Fig. 5.—Bronchial foreign bodies: (1) Coffee bean, male, 6 months and 24 hours' duration. (2) Metal tube, female, 11 months and 24 hours. (3) Porcelain, male, 2 years and 1 month. (4) Piece of celluloid toy, male, 1 year and 5 hours. (5) Watermellon seed, male, 12 months and 17 days. (6) Piece of match, male, 29 years and 3 days. (7) Corn kernel, female, 2 years and 60 hours. (8) Human tooth, female, 12 years and 4½ months. (9) Screw, male, 3½ years and 13 months. (10) Tin foil, male, 8 years and 4 days. (11) Navy bean, male, 15 months and 5 hours. (12) Nail, male, 3 years and 4 hours. (13) Nail, female, 5 years and 38 hours. (14) Pencil top, female, 12 years and 15 hours. (15) Peanut shell, female, 16 months and 8 hours. (16) Peanut, female, 15 months and 24 hours. (17) Russian peanut husk, male, 20 months and 3 days. (18) Almond husk, male, 16 years and 24 hours. (19) Pork bone, male, 11 months and 24 hours. (20) Chicken bone, male, 21 months and 20 hours. (21) Wax crayon, male, 22 months and 9 days.

esophagoscopy, and three died in the postoperative period of pneumonia, acute laryngotracheobronchitis, and malignancy. Of these five deaths, two were due to the endoscopic procedure. The over-all mortality rate was 0.8%.

Comment

Bronchoesophagology, primarily as a means for removal of foreign particles from the respiratory passages, received its impetus in about 1895 through the work of Killian.† He removed a piece of bone from the bronchial tree with a bronchoscope, through a tracheotomy wound. Prior to that time foreign bodies in the respiratory passages were either fatal or were treated by bronchotomy, which was attended by 23% mortality. Mackenzie is reported to have used a skeleton type of esophagoscope as early as 1890. Before that foreign particles in the esophagus were treated with various probes, bougies, and hooks, with some success.

It was not until about 1905, however, that general usage of bronchoscopy and esophagoscopy for recovery of foreign particles in the air and food passages was promoted to a useful technique by Chevalier Jackson. In the succeeding 20 years foreign-body endoscopy was advanced from a medical curiosity to an exacting technique.‡ With time, as endoscopists became skilled in these techniques, it was recognized more and more that the peroral endoscopy had a diagnostic as well as a therapeutic role.§

In recent years bronchoesophagology as a diagnostic measure has largely surpassed the foreign-body work in frequency.‖ Yet, foreign bodies are still far from rare, and often the obstructed airway depends upon the skill of the endoscopist for removal of the intruding particle.¶ Even with the advancing skill of the chest surgeon in the removal of a lung or a segment thereof or in esophageal surgery, it is still preferable to recover foreign material from the lung or the esophagus by endoscopic procedures. For this reason it is highly unlikely that the fine skill of the expert endoscopist will ever be replaced.

One of the true remaining emergencies in medicine today is the problem of the obstructed airway. Although the majority of patients with foreign particles in the air and food passages have only mild to moderate obstruction to respiration, occasionally there will be severe embarrassment, which will result in the patient's death unless relief is immediate. In these patients, the burden of treatment rests on the physician who first sees the patient. If the respiratory obstruction is in the glottis or above and the obstructing element cannot be removed immediately with the finger or some instrument, tracheotomy is mandatory. The for-

Fig. 6.—Lateral roentgenogram of the throat showing chicken bone in the cricopharyngeal area.

† References 5 and 6.
‡ References 7-25.
§ References 26-33.
‖ References 34-43.
¶ References 44-49.

eign particle can be removed at leisure when the airway has been established. If the foreign particle is located in the esophagus or the bronchial tree, then the services of an expert bronchoesophagologist are needed.

In many cases, the possibility of a foreign particle in the air and food passages is suspected but cannot be established by direct examination. This can be confirmed in many instances by radiological evaluation of the patient. In patients with suspected upper esophageal obstruction due to a foreign particle, many radiopaque objects will be revealed by posteroanterior and lateral views of the throat and chest. If the particle has slipped into the chest and is nonopaque, there is less chance of demonstration by direct x-ray. In these cases, the particle or the obstruction level may be seen after the ingestion of contrast medium.

Fig. 7.—Oblique view of the chest showing open safety pin in the midesophagus.

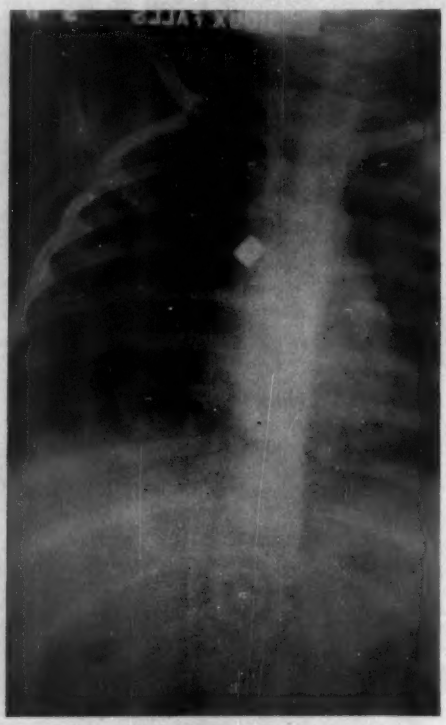

Fig. 8.—Metal cylinder in the right main bronchus of an 11-month-old child.

Foreign particles in the lung are visualized radiographically by several means. These include the following: 1. Direct visualization of the particle with posteroanterior and lateral views (possibly oblique film also) in the case of radiopaque material. 2. The presence of hyperventilation, as is seen on inspiratory and expiratory films of the chest, suggests the presence of bronchial obstruction. Unilateral hyperventilation indicates a ball-valve type of obstruction on the affected side, possibly by extraneous material. 3. Tracheal shift usually indicates the presence of bronchial obstruction but may be confusing in the diagnosis of foreign body in the lung. Soon after inhalation, due to the ball-valve effect, the shift may be to the opposite side. Later, after atelectasis has developed, the shift will be to the affected side. 4. Atelectasis of a lung or a segment thereof indicates

bronchial obstruction which may be due to a foreign particle which has been in place for some time. Although atelectasis can develop in a matter of a few hours, it is usually seen best in bronchial occlusion by a particle for a longer period of time. Unexplained pneumonitis or atelectasis in a child must always be suspected of exogenous origin. 5. Fluoroscopic examination of the throat and chest, with or without the

Fig. 9.—Localized atelectasis, right upper lobe, secondary to bronchial obstruction by peanut.

use of contrast media, may be very helpful in questionable cases.

In any case in which there is possibility of foreign material in the esophagus or the bronchial tree, careful radiological evaluation is indicated. Endoscopic examination is indicated if ingested or inhaled foreign body is suspected from the history or the physical findings, even though the x-rays may be entirely negative. Figures 6, 7, 8, 9, and 10 all show typical x-ray findings in patients from this series with foreign bodies in the tracheobronchial and esophageal passages.

Fig. 10.—Localizing x-ray taken during bronchoscopy showing screw in right main bronchus, bronchoscope, and foreign-body forceps.

Summary

During the 33-year period from 1921 until 1954, five hundred twenty-three endoscopic examinations were performed in Sioux Falls by one otolaryngologist. There were 370 foreign bodies found in the air and food passages, 24 malignant neoplasms were diagnosed, 11 benign neoplasms were found and treated, and 61 other pathological processes were noted. Fifty-three examinations were negative for foreign body or other specific pathology. The majority of the foreign particles in the air and food passages were found in children, and, as would be expected, the majority of the neoplasms were found in adults. The find-

ings suggest that the fine art of foreign-body endoscopy is very much in need in a community of this size. Diagnostic bronchoscopy and esophagoscopy have recently become an increasingly important tool in the study of patients suspected of neoplasm, stricture, congenital anomaly, inflammatory processes, and varices in the air and food passages. The preoperative work-up and the postoperative care of the chest-surgery patients depend increasingly upon the skill of a competent endoscopist.

Although the hazards of peroral endoscopy have been reduced to essentially nil by the use of antibiotic agents, refinements in technique, and the development of new instruments, these procedures should not be undertaken inadvisedly. In the hands of the skilled endoscopist, bronchoscopy, esophagoscopy, and laryngoscopy have contributed much to this community and to the advancement of medicine in this vicinity. There is no reason why these diagnostic and therapeutic tools cannot continue to perform a valuable service in the hands of thoroughly trained and skilled endoscopists.

14 Riverview Heights.

REFERENCES

1. Barrett, J. H.: Foreign Bodies in the Air and Food Passages, A. M.A. Arch. Otolaryng. 54:651-665 (Dec.) 1951.

2. Boyd, G.: Oesophageal Foreign Bodies, Canad. M. A. J. 64:102-107 (Feb.) 1951.

3. Grekin, T. D., and Musselman, M. M.: The Management of Foreign Bodies in the Alimentary Tract, Ann. Surg. 135:528-535 (April) 1952.

4. Burton, W. L.: Per-Oral Endoscopy, J. M.A. Georgia 1:35-38 (June) 1947.

5. Clerf, L. H.: Historical Aspects of Foreign Bodies in the Air and Food Passages, Tr. & Stud. Coll. Physicians Philadelphia 20:10-16 (June) 1952.

6. Famous Firsts: The First Bronchoscope, Therapeutic Notes, Parke Davis & Company, 62: 57 (Feb.) 1955.

7. Jackson, C.: Bronchoscopy and Esophagoscopy: A Brief Consideration of Technique, Surg. Gynec. & Obst. 44:795-804 (June) 1927.

8. Clerf, L. H.: Peroral Endoscopy, Arch. Otolaryng. 9:318-342 (March) 1929; 11:343-360 (March) 1930; 13:444-461 (March) 1931.

9. Jackson, C. L.; Tucker, G.; Clerf, L. H.; Lukens, R. M., and Moore, W. F.: Bronchoscopy as an Aid to the Thoracic Surgeon, J.A.M. A. 84:97-102 (Jan. 10) 1925.

10. Jackson, C.: Foreign Bodies in the Air and Food Passages, Reprinted from the Transactions of the American Laryngological, Rhinological and Otological Society, Reynolds, the Printer, New Bedford, Mass., 1924.

11. Naftzger, J. B.: Stridor and Dyspnea in Childhood, J. Iowa M. Soc. 13:367-371 (Sept.) 1923.

12. Gittins, T. R.: Foreign Bodies in the Air and Food Passages, J. Iowa M. Soc. 15:532-537 (Oct.) 1925.

13. Gittins, T. R.: Peanut in the Bronchus of an Infant Aged 22 Months, Arch. Otolaryng. 11: 614-615 (May) 1930.

14. Gittins, T. R.: Foreign Bodies in the Air and Food Passages: Observations in 81 Cases, with Special Reference to Overlooked Foreign Bodies, Ann. Otol. Rhin. & Laryng. 40:183-198 (March) 1931.

15. Myerson, M. C.: What Children Put in Their Mouths, Hygeia 10:216-218 (March) 1932.

16. Clerf, L. H.: Foreign Bodies in the Air and Food Passages: Observations on a Series of 50 Cases, Laryngoscope 34:784-810 (Oct.) 1924.

17. Jackson, C.: Bronchoscopic Cases of Dental Interest: Dental Foreign Bodies in the Air and Food Passages, J. Am. Dent. A. 14:1341-1370 (Aug.) 1927.

18. Pitkin, C. E.: Foreign Bodies of Dental Origin in the Air and Food Passages, J. Am. Dent. 11:275-285 (March) 1924.

19. Moore, W. F., and Lukens, R. M.: The Bronchoscopic Treatment of Pulmonary Suppurations, and Bronchial Asthma, Ann. Otol. Rhin. & Laryng. 33:956-957 (Sept.) 1924.

20. Flick, J. B.; Clerf, L. H.; Funk, E. H., and Farrell, J. T.: Pulmonary Abscess, Arch. Surg. 19:1292-1312 (Dec.) 1929.

21. Jackson, C. L.: Chronic Nonspecific Infections of the Lungs, J.A.M.A. 87:729-736 (Sept. 4) 1926.

22. Tucker, G.: Retropharyngeal Abscess from Lodgment of Foreign Body in Esophagus, J.A. M.A. 84:511-512 (Feb. 14) 1925.

23. Tucker, G.: Roentgenologic Aid to the Bronchoscopist, Arch. Phys. Therap. 7:571-583 (Oct.) 1926.

24. Dean, L. W., and Gregg, J. B.: An Implantation Cyst of the Larynx, Laryngoscope 28:74-77 (Feb.) 1918.

25. Gregg, J. B.: The Practical Application of Endoscopy, Journal-Lancet 43:485-488 (Oct. 1) 1923.

26. Manges, W. F., and Clerf, L. H.: Congenital Anomalies of the Alimentary Tract, Am. J. Roentgenol. 33:657-669 (May) 1935.

27. Symposium on Respiratory Obstruction in

Infancy and Childhood, Proc. Staff Meet. Mayo Clin. 25:345-376 (June 21) 1950.

28. Symposium on Bronchogenic Carcinoma, Proc. Staff Meet. Mayo Clin. 19:353-376 (July 12) 1944.

29. Clerf, L. H.: Bronchoscopy in Bronchopulmonary Disease, Mod. Med. 13:53-60 (April) 1945.

30. Holinger, P. H.: Indications for Bronchoscopy in Pulmonary Disease, Journal-Lancet 65: 235-238 (July) 1945.

31. Hoskins, H. D.: Indications for Broncho-Esophagology, Virginia M. Month. 75:182-184 (April) 1948.

32. Waterman, D. H.: Bronchoscopy: Its Indications and Value, J. Tennessee M. A. 39:406-413 (Nov.) 1946.

33. Iglauer, S.: Bronchoscopy as a Diagnostic and Therapeutic Procedure, Nebraska M. J. 28: 340-344 (Nov.) 1943.

34. Clerf, L. H.: Progress in Bronchology, J.A.M.A. 136:733-736 (March 13) 1948.

35. Ochsner, A.; DeBakey, M. E., and Dixon, J. L.: Primary Malignancy of the Lung, South Dakota J. Med. & Pharm. 1:43-47 (Feb.) 1948.

36. Ochsner, A.: Bronchogenic Carcinoma: The Role of the Bronchoscopist in Its Early Diagnosis, Selected Writings from the Ochsner Clinic, The Alton Ochsner Medical Foundation Publication, New Orleans, 12:9-16 (Dec. 31) 1953.

37. Symposium on Certain Tumors of the Bronchi (Adenomas and Cylindromas) and Tumors of the Trachea, Proc. Staff Meet. Mayo Clin. 21:409-432 (Oct. 30) 1946.

38. Dean, L. W., and Gregg, J. B.: Report of a Case of Coexistent Carcinoma, Tuberculosis, and Syphilis of the Esophagus, Ann. Otol. Rhin. & Laryng. 26:619-632 (Sept.) 1917.

39. Gregg, J. B.: The Prognosis and Treatment in Tuberculosis of the Larynx, Journal-Lancet. 41:87 (Sept. 15) 1921.

40. Gregg, J. B.: The Prognosis of Tuberculous Larynx, Ann. Otol. Rhin. & Laryng. 30:1007-1050 (Dec.) 1921.

41. Gregg, J. B., and Stamler, F. W.: Unusual Neoplasms of the Esophagus, A.M.A. Arch. Otolaryng. 59:159-169 (Feb.) 1954.

42. Gregg, J. B.: Lung Neoplasms from the Endoscopist's Standpoint, South Dakota J. Med. & Pharm. 7:203-209 (July) 1954.

43. Gregg, J. B.; Merkel, B. M., and Cross, K. R.: Endobronchial Placer Mining for Neoplastic Cells, A.M.A. Arch. Otolaryng. 61:267-276 (March) 1955.

44. Bunker, P. G.: Vegetable Foreign Bodies in the Tracheobronchial Tree, Journal-Lancet 64: 359-374 (Nov.) 1944.

45. Bunker, P. G.: Method of Rotating the Open Safety Pin in the Esophagus, Arch. Otolaryng. 37:78-81 (Jan.) 1943.

46. Phelps, K. A.: Foreign Bodies Lodged in the Air or Food Passages, Minnesota Med. 27: 27-31 (Jan.) 1944.

47. Clerf, L. H.: Foreign Bodies in Air and Food Passages: Observations on End Results in Series of 950 Cases, Surg. Gynec. & Obst. 70: 328-340 (Feb.) 1940.

48. Equen, M.: Magnetic Removal of Foreign Bodies from the Food and Air Passages, Arch. Otolaryng. 46:225-229 (Aug.) 1947.

49. Equen, M.; Roach, G.; Brown, R., and Bennett, T.: Open Safety Pin in the Esophagus of a Septuagenarian, A.M.A. Arch. Otolaryng. 62:426-428 (Oct.) 1955.

Osteomyelitis of the Frontal Bone

Cumulative Information, X-Ray Interpretations, and a Study of Nine Selected Cases of Cranial Disease

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Perhaps my attitude toward osteomyelitis of the frontal bone has been too casual. In any case, I did not report three interesting cases of frontal osteitis that came to my attention four years ago; and I did not publish a series of such occurrences that had been under my observation for a long time.

These reflections, prompted by the analysis of some 35 references of osteomyelitis of the frontal bone, force me to recognize that my association with this disease—at least in a comparative way—has been more extensive than I had realized.

With all due respect to Drs. Mosher, Fred, Judd, Furstenberg, Maxwell, Bulson, Williams, Morrison, and others, I challenge anyone to read their dissertations on osteomyelitis of the frontal bone without sensing a certain feeling of inadequacy. Also, I have observed that our knowledge of "osteo" of the frontal bone has not progressed as has our know-how of "osteo" of the mastoid bone.

Emerson said: "Progress is an unfolding like a vegetable bud. You first have an instinct, then an opinion and then a knowledge."

According to my experience, "osteo" of the frontal bone is in the process of passing from the opinion stage into the knowledge stage.

If one accepts the maxim that knowledge is the cumulative thought and experience of innumerable minds, then the bibliography on osteomyelitis of the frontal bone is the essence of our present understanding concerning it. But is this knowledge or understanding complete? The answer is "No"!

How simple were the patent-medicine days when wealth and impoverishment were the result of such slogans as the following:

"Magill's pills cure all ills. If they do not do it, he pays the bills!"

But we must be practicable. Shall we pass over the controversial points so much discussed by our authorities on osteomyelitis of the frontal bone and consider only those items upon which they agree or compromise their ideas?

Here, then, are some of the basic thoughts from our cumulative knowledge of osteomyelitis of the frontal bone which continue to guide us.

Following a prescribed pattern, these particulars are discussed as they have been presented and under the following heads:

1. Type
2. Etiology
3. Signs and Symptoms
4. Diagnosis
5. Pathology
6. Treatment
7. Complications
8. Bacteriology
9. Radiology

1. Types

There are three distinct varieties of osteomyelitis of the frontal bone, namely: (*a*) the fulminating, (*b*) the acute, and (*c*) the chronic.

The fulminating form (and the most dreaded of the three) is apparently diminishing under present-day management.

The acute form occurs as a complication of a frontal-sinus infection, but, more spe-

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cifically, it tends to follow surgery on a frontal sinus that is acutely inflammed.

The chronic form is a quiet continuation of an acute phase which has been inadequately or improperly managed or which has not responded even to the most accepted form of treatment.

2. Etiology

(*a*) May I repeat that "acute frontal sinusitis followed by frontal sinus surgery is the most common cause of Osteomyelitis of the frontal bone"?

(*b*) Intranasal operations in the presence of acute frontal-sinus manifestations are frequent causes of "osteo" of the frontal bone.

(*c*) Trauma (accidental or operative) is an important etiologic factor.

(*d*) Underlying all these causes is pus under pressure in an acutely infected frontal sinus.

3. Signs and Symptoms

(*a*) Forehead swelling in the presence of an acute frontal sinusitis, with or without a history of accidental or operative trauma, is almost pathognomonic of "osteo" of the frontal bone.

(*b*) Often the patient's general condition is unaffected.

(*c*) Variant headaches and fever are common, but the appearance of intracranial manifestations clinches the diagnosis of "osteo" of the frontal bone.

4. Diagnosis

The signs and symptoms as already mentioned plus x-ray evidence of osteitic bone reaction confirm the nature of the disease.

5. Pathology

The flat bones of the cranium consist of three layers: (*a*) the external table, which is compact bone; (*b*) the internal table, which is compact bone, and (*c*) the intervening diploetic structure, which is spongy bone.

It is the infection within the spongy bone that kindles and diffuses the osteomyelitis. "The veins of the diploe are an irregular network of unwalled spaces that lead: (*a*) to the periosteum; (*b*) to the dura; and (*c*) to the soft tissues."

Acute "osteo" of the frontal bone is originally a thrombophlebitis of the veins of the frontal-sinus mucosa which drain directly into the diploetic spaces of the skull. Subacute "osteo" and chronic "osteo" are bony necroses or sequestrations resulting from a frontal-bone thrombosis or endarteritis that shuts off the blood supply to an affected part. Histologically one cannot distinguish between an acute and chronic "osteo."

Uniformly osteomyelitis of the frontal bone appears to originate at the point at which the anterior and posterior walls of the frontal sinuses converge, and in a position comparable to the upper internal corner.

Interesting, too, is the observation that the large diploetic veins of the cranium pass from bone to bone regardless of intervening suture lines—a condition that enables thrombi and emboli to carry infection over apparently normal tissue to more distant parts of the skull.

In my experience I have never observed a primary osteomyelitis of the frontal bone, nor am I convinced that there is such a condition as a hematogenic frontal osteitis.

My cases of osteomyelitis of the frontal bone seem always to reduce themselves to one basic equation, namely: acute frontal sinusitis + trauma (accidental or operative) = an "osteo" of the frontal bone.

And yet one wonders why frontal-bone osteitis is so infrequent in its occurrence!

I shall not discuss the manner in which frontal-sinus surgery in the presence of an acute frontal sinusitis breaks down nature's barrier against an anterior cranial osteomyelitis, for I am not sure what those barriers are, nor am I sure they exist.

6. Treatment

When a fulminating frontal sinusitis gets out of bounds and invades the anterior cranium, the patient's condition is always critical and presents a situation requiring prompt and corrective measures that are adequate and safe. But what measures are prompt and corrective and adequate and safe?

In my opinion, intranasal surgery under

such circumstances is always hazardous and contraindicated. Likewise, I have observed that even a chiselling of the diploetic anterior frontal-sinus wall may be a risky piece of business. I agree with many of our authorities that the floor of the frontal sinus, which is compact bone (almost devoid of diploetic structure), is, at least in theory, the preferred locale for surgical drainage, but for reasons of intraorbital extension, I hesitate to use it.

In any case, a fulminating "osteo" of the frontal bone requires the best of medical and surgical judgment. Only one's experience can guide him in the proper decisions and actions.

If and when the acute manifestations of frontal "osteo" subside, effectual surgery can be performed; but one cannot always wait for such a subsidence. Incidentally, any form of sinus or cranial surgery should be combined with the most responsive type of antibiotic therapy.

7. Complications

In the wake of the pathological processes of osteomyelitis of the frontal bone come the complications, many of which are terminal affairs: The common ones are (*a*) meningitis, (*b*) epidural abscess, (*c*) brain abscess, (*d*) thrombosis of the venous sinuses, (*e*) epileptiform contractions, and (*f*) hemiplegia.

8. Bacteriology

So far as I know, bacteriological studies have added nothing to our knowledge of osteomyelitis of the frontal bone.

9. Radiology

I cannot conceive of diagnosing or managing a case of "osteo" of the frontal bone without the constant assistance of radiography.

My entree to the present investigation of osteomyelitis of the frontal bone was by way of the x-ray department.

In a study of nine selected cases I shall present an abstracted account of the x-ray interpretations of certain cranial manifestations and in each case compare the x-ray interpretation with its clinical history and operative or postmortem findings.

I shall also make a comment concerning the manner in which each case was or was not consistent with cumulative information on the subject.

Summary of the Selected Cases of Cranial Disease

Case Number	Age	Sex	Year Last Observed	Confirmed Diagnosis	Outcome
1	52	F	1955	"Osteo"	Deceased
2	52	M	1954	Malignancy	Deceased*
3	63	M	1952	"Osteo"	Well
4	47	M	1952	"Osteo"	Well (fistula)
5	66	F	1951	Mucocele	Well*
6	17	M	1941	"Osteo"	Deceased
7	33	F	1935	"Osteo"	Deceased
8	30	F	1932	"Osteo"	Deceased
9	13	F	1932	"Osteo"	Deceased

* The malignancy and mucocele cases were included in this study because the x-ray interpretations were indicative of an "osteo" of the skull.

A Study of Selected Cases

CASE 1.—A woman, aged 52 years, saw her doctor for the first time two weeks before her death. She gave a history of a stuffy nose, headaches, and a dacryocystitis that had ruptured externally. None of these symptoms were serious. Three weeks later she had a chill, a fever of 105 F, and a stiff neck.

X-rays showed an "osteo" of the right frontal bone with extensive sequestration.

A conservative external frontal-sinus operation revealed an empty cavity but a perforated posterior wall. At operation a bronchoscopy and tracheotomy were required because the main bronchi were filled with mucous secretion.

Five days later the patient died. Autopsy revealed an "osteo" of the frontal bone, a diffuse purulent meningitis, an epidural abscess, a primary atelectasis, and a hydrothorax.

Comment.—The x-ray diagnosis was confirmed.

CASE 2.—A man, aged 52, had a history of repeated polypectomies performed over a period of 20 years. For 10 or 12 years he had a recognized inoperable squamous-cell cancer of the frontal and ethmoidal sinuses. At least every 6 to 12 months

Fig. 1 (Case 1).—X-ray diagnosis: "Osteo of the right frontal bone with extensive sequestration." Clinical diagnosis: "Osteo of the frontal bone, diffuse purulent meningitis, epidural abscess, and primary atelectasis and hydrothorax."

the frontal sinuses and ethmoidal cavities were exenterated to relieve a proptosis and to give him more breathing space.

The original x-ray diagnosis was "destructive bone change indicative of Osteo of the frontal bone."

In October of 1951 an extensive frontal craniotomy confirmed the diagnosis of an inoperable carcinomatous extension into both orbits.

Approximately two and one-half years later the man died. Autopsy revealed a diffuse meningitis, a left frontal-lobe abscess (draining through the frontal defect), encephalitis, and a terminal pneumonia.

Comment.—There was no true "osteo" of the frontal bone. The x-ray diagnosis of "destructive bone change indicative of Osteo of the frontal bone" was not confirmed.

Case 3.—A man, aged 63, gave a history of a nasal polypectomy 12 years before his admission to the hospital. Three days prior to his recent difficulty he developed a cellulitis over the left eye. After an attempted irrigation of the left frontal sinus he developed a headache and a fever of 102 F.

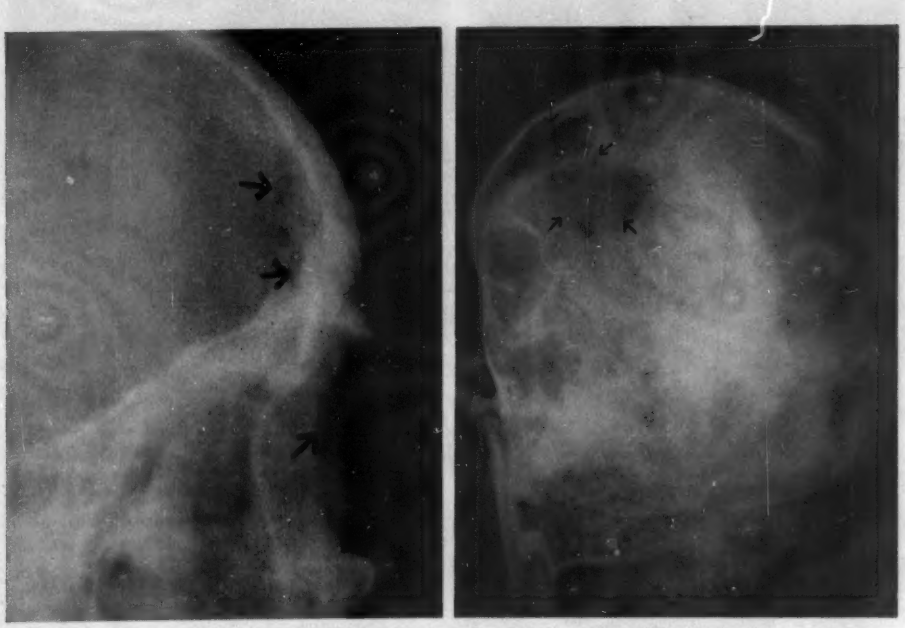

Fig. 2 (Case 2).—X-ray diagnosis: Preoperative interpretation: "Destructive bone change indicative of Osteo of the frontal bone." Postoperative interpretation: "Operative bone defect—? Osteo or malignancy." Clinical diagnosis: "Extensive malignancy of frontal and ethmoid sinuses and orbits—large operative defect—No Osteo of Skull—Meningitis, frontal lobe abscess, encephalitis and terminal pneumonia."

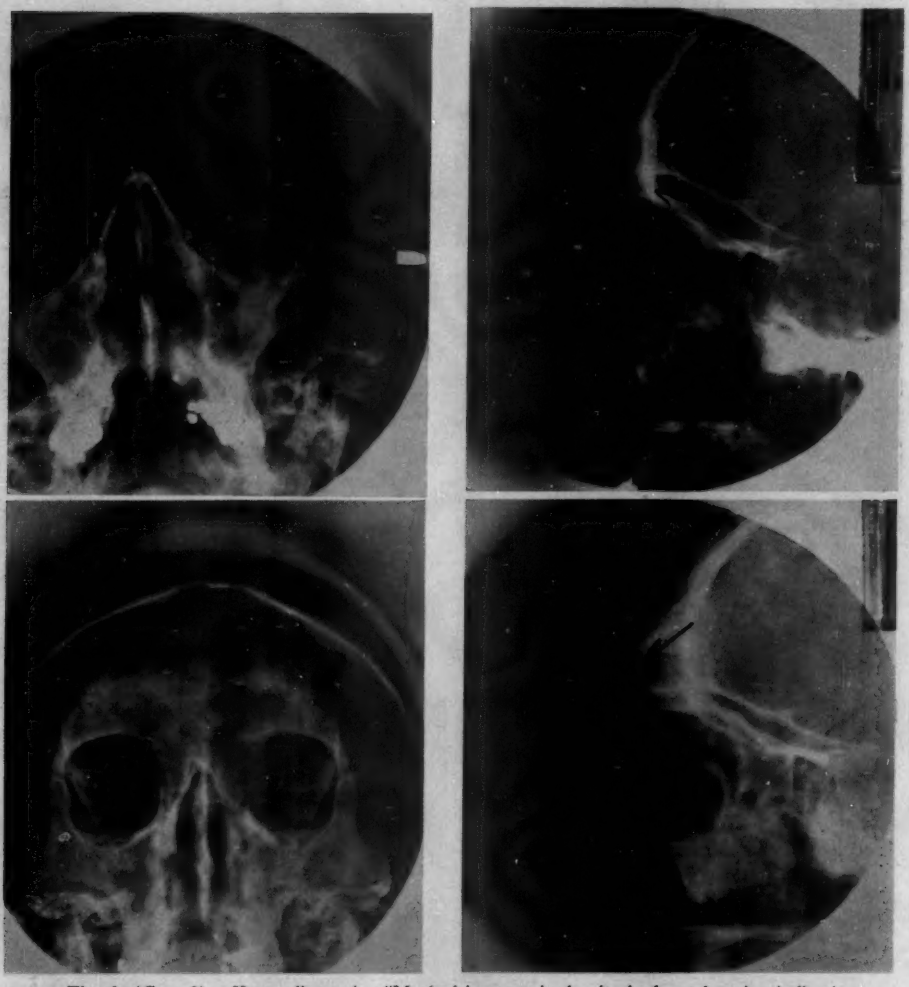

Fig. 3. (Case 3).—X-ray diagnosis: "Marked increase in density in frontal region indicative of Osteo." Clinical diagnosis: "Osteo of the frontal bone."

The x-ray diagnosis was "Marked increase in density in the frontal region."

A radical frontoethmoidectomy was performed. Ten days later there was recurring frontal edema. Because the patient refused further sinus surgery, an interval of one month elapsed, during which time a diagnosis of "clinical osteo" was made. With the performing of a frontal craniotomy and the insertion of a Vitallium plate the patient "got well."

Comment.—Attention should be directed to the intranasal probing in an acute frontal sinus preceeding "osteo," to the unsuccessful frontal ethmoidectomy, and to the successful craniotomy.

Case 4.—A man, aged 47, presented a history of a purulent frontal sinusitis for 20 years with numerous episodes of frontal swelling, headaches, and spontaneous drainages through an external sinus fistula. He also had arrested advanced pulmonary TB and "adequately treated" CNS syphilis.

Repeated x-ray diagnoses were "Extensive reactive bone change throughout the frontal area and sequestration indicative of Osteo."

Repeated frontal craniotomies and sequestrectomies confirmed the x-ray diagnosis of chronic "osteo" of the frontal bone.

Fig. 4 (Case 4).—X-ray diagnosis: "Extensive reactive bone change through frontal area—sequestration (right) indicative of Osteo." Clinical diagnosis: "Osteo of the frontal bone."

Comment.—When last seen, in 1952, this man was vomiting blood from activated advanced pulmonary TB and the "osteo" was still draining through an external fistula.

CASE 5.—A woman, aged 66, was referred to the x-ray department by her oculist. She complained of recurrent swellings of the right eye for 10 years. There was no history of pain or nasal discharge.

The x-ray diagnosis: "Osteomyelitis of the frontal bone with erosion into the orbit."

Operation revealed a large mucocele with a massive area of dural exposure.

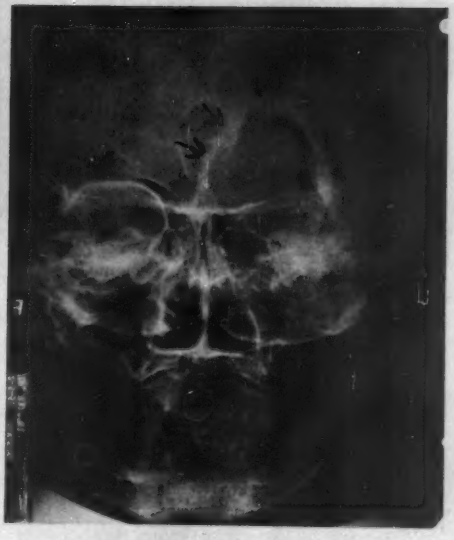

Fig. 5 (Case 5).—X-ray diagnosis: "Osteo of the frontal bone." Clinical diagnosis: "Large mucocele with a massive area of dural exposure."

Fig. 6 (Case 6).—X-ray diagnosis: "? of marginal Osteo." Clinical diagnosis: "Osteo of the right frontal bone."

Comment.—The operation proved the x-ray diagnosis of "osteomyelitis of the frontal bone" to be incorrect. The patient had a pressure necrosis in the roof of the orbit from the long-continued expanding mucocele. When last seen, in 1951, this patient was well.

Case 6.—A boy, aged 17 years, gave a history of having sustained a blow on the forehead from the butt of a small gun when he was 10 years old. At that time he first came to a hospital because "he felt sick in the head." X-rays revealed no evidence of fracture or other cranial pathology.

Seven years later he came to the hospital, again, because he "felt sick in the head"—a symptom of 10 days' duration. The clinical diagnosis was "acute right frontal sinusitis with orbital abscess."

X-rays revealed a right frontal, ethmoidal, and maxillary sinusitis, with no evidence of "osteo" of the skull.

X-ray interpretations made a week later read, "It was not possible to establish a definite diagnosis of Osteo of the Skull but it may have existed."

No operation was performed. Eleven days after entering the hospital the patient died.

Autopsy findings were "supra-orbital sinusitis with Osteo of the right frontal bone, a frontal brain abscess and terminal pneumonia."

Comment.—The x-ray interpretations in this instance were inconclusive, but the case proved to be one of a fulminating "osteo."

Fig. 7 (Case 7).—X-ray diagnosis: "Bilateral reactive bone change in frontal area, indicative of chronic Osteo." Clinical diagnosis: "Osteo of the frontal bone."

Case 7.—A woman, aged 33, presented a history of long-standing nasal discharge and headaches, for which she had received treatment for many years.

The x-ray diagnosis was "bilateral maxillary and ethmoidal sinusitis."

Bilateral ethmoidectomies, bilateral antrum windows, and a submucous resection were followed later by a right radical antrum operation, bilateral

turbinectomies, and bilateral intranasal sinusotomies.

Later she underwent bilateral external sinusotomies, at which time there were signs and symptoms of meningitis. Two weeks subsequently a craniotomy revealed an "osteo" of the frontal bone and a right frontal-lobe abscess.

Fig. 8 (Case 8).—X-ray diagnosis: "Early destructive changes in margins of right frontal sinuses, indicative of Osteo." Clinical diagnosis: "Osteo of the skull."

The following day (after the operation) the patient died. No autopsy was performed. The x-ray diagnosis of "osteo" of the frontal bone was confirmed at operation.

Comment.—Were there too many pre-osteomyelitis operations?

CASE 8.—A woman, aged 30, complained of sinus trouble for nine weeks. Her chief complaint was neuralgic head pain.

The x-ray diagnosis was "early destructive changes in the margins of the right frontal sinus, indicative of Osteo."

A right external frontal sinus gave her no relief. The provisional diagnoses on entering the hospital were a right frontal sinusitis, "osteo" of the right frontal bone, and multiple brain abscesses.

A frontal craniotomy revealed an "osteo" of the frontal bone and right frontal-lobe abscesses.

Two weeks later the patient died. Autopsy revealed a subdural abscess, "osteo" of the skull, focal encephalitis, a herniation of the frontal lobe through the operative defect, and a terminal pneumonia.

Comment.—The x-ray diagnosis of "osteo" of the frontal bone was confirmed at operation. The "osteo" was of the fulminating type.

CASE 9.—A girl, 13 years of age, came into the hospital with a headache, stiff neck, and a temperature of 105 F following removal of turbinates and

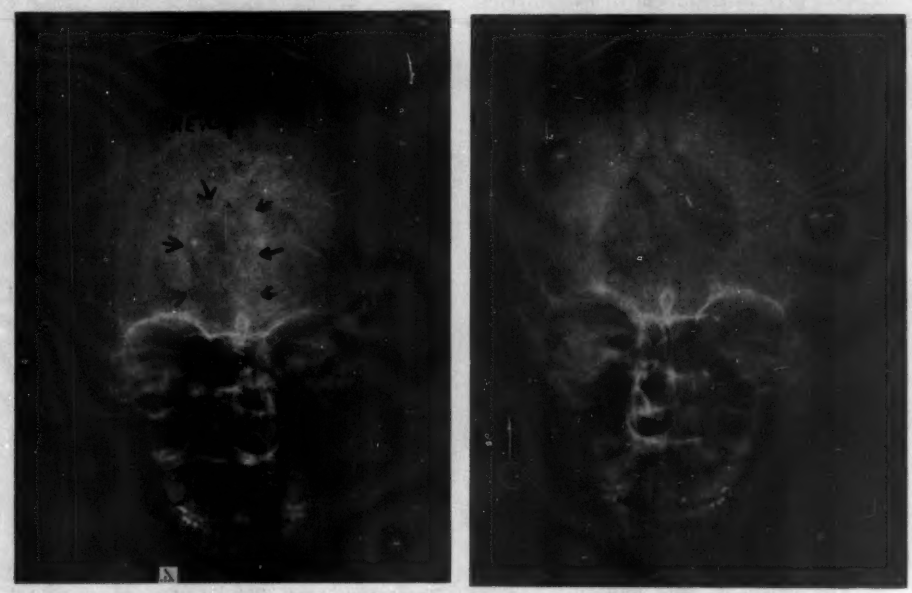

Fig. 9 (Case 9).—X-ray diagnosis: "Osteo of the frontal bone." Clinical diagnosis: "Osteo of the frontal bone."

several intranasal frontal-sinus operations for the relief of an acute frontal sinusitis.

No x-rays were taken before these operations. Provisional diagnoses of "Pan-sinusitis, Osteo of the frontal bone, and a question of a frontal lobe abscess" were made.

When the patient was admitted to the hospital the left side of the forehead was bulging. X-rays showed "findings that suggest congenital changes perhaps due to blood vessels such as a hemangioma of the meninges pressing against the inner table of the frontal bone; calcification of the choroid plexus; Osteo of the skull does not resemble this picture."

A month later external drainage of the frontal sinuses revealed a dehiscence of the internal plate, with a thickening of the adjacent dura. A subsequent frontal craniotomy resulted in fairly good recovery, but after a period of two months the patient had projectile vomiting and headaches. Reoperation disclosed a deep-seated abscess in the right frontal lobe. Within a month the patient died. Autopsy revealed a pansinusitis, an "osteo" of the frontal bone, a right frontal-lobe abscess, a subacute meningitis, and an encephalitis. These findings confirmed the x-ray diagnosis.

Comment.—This case raises the question of the advisability of performing intranasal operations during acute frontal sinusitis.

Conclusions

1. Our present treatment of osteomyelitis of the frontal bone is fundamentally the same as it has been for many years.

2. The use of antibiotics is often helpful, but it is not a specific form of therapy.

3. Nasal and paranasal surgery procedures in the presence of an acute frontal sinusitis are often followed by frontal osteitis.

4. Operation on an acute or fulminating "osteo" of the frontal bone may lead to fatal intracranial complications.

5. Failure to operate on an acute or fulminating "osteo" of the frontal bone may also lead to fatal intracranial complications.

6. Chronic "osteo" of the frontal bone is usually mildly progressive and subject to fulminating exacerbations.

7. Repeated x-ray interpretations of the skull are essential to the management of all cranial diseases. Only rarely are the interpretations incorrect or indecisive.

8. The morbidity and mortality rate in "osteo" of the frontal bone is still too high.

Bibliography

Bulson, A. E., Jr.: Osteomyelitis of the Frontal Bone as a Complication of Frontal Sinusitis, J. A. M. A. 86:246-250 (Jan. 23) 1926.

Cirillo, A. A.: Frontal Osteomyelitis with Subdural Abscess and Hemiplegia After Frontal Sinusitis, J. A. M. A. 121:105-107 (May 11) 1946.

Dill, J. L.: Osteomyelitis of the Frontal Bone, Laryngoscope 52:267-274 (April) 1942.

Fred, G. B.: Acute Fulminating Osteomyelitis of Frontal Bone: A Review and a Prediction, Ann. Otol. Rhin. & Laryng. 51:638-647 (Sept.) 1942.

Furstenberg, A. C.: The Pathology of the Spread of Osteomyelitis of the Skull: Its Relation to Brain Abscess, Tr. Am. Laryng. Rhin. & Otol. Soc. 39: 423-429, 1933.

Osteomyelitis of the Skull: The Osteogenetic Processes in the Repair of Cranial Defects, Ann. Otol. Rhin. & Laryng. 40:996-1012 (Dec.) 1941.

Galloway, T. C.: Infections with Anaerobic Streptococci, with Special Reference to Cranial Osteomyelitis, Arch. Otolaryng. 36:514-525 (Oct.) 1942.

Glass, G.: Case of Osteomyelitis of Frontal Bone Following Acute Frontal Sinusitis, Treated by Surgery and Penicillin, J. Laryng. & Otol. 62:310-313 (May) 1948.

Hardy, A. N.: Osteomyelitis of the Frontal Bone with Brain Abscess, Canad. M. A. J. 54:374-376 (April) 1946.

Herberts, G.: Frontal Osteomyelitis: Its Treatment and Some Experimental Observations on the Effect of Heparin in Combination with Sulphonamide and Penicillin Therapy, Acta oto-laryng. 37: 321-333 (Aug.) 1949.

Kirby, W., and Hepp, V. E.: Treatment of Osteomyelitis of the Facial Bones with Penicillin, J. A. M. A. 125:1019-1022 (Aug. 12) 1944.

Kos, C. M.: Microscopic Changes in Osteomyelitis of the Frontal Bone, Ann. Otol. Rhin. & Laryng. 49:510-513 (June) 1940.

Lillie, H. L.: Osteomyelitis of the Cranial Bones Secondary to Paranasal Sinus Operations, Ann. Otol. Rhin. & Laryng. 34:353-360 (June) 1925.

Macbeth, R. G.: Osteomyelitis of the Frontal Bone of Rhinogenic Origin, Proc. Roy. Soc. Med. 42:284-294 (May) 1949.

Maxwell, J. H.: Chronic Proliferative Osteomyelitis of the Skull, Ann. Otol. Rhin. & Laryng. 55:719-742 (Dec.) 1946.

McKenzie, D.: Diffuse Osteomyelitis from Nasal Sinus Suppuration, J. Laryng. & Otol. 28:6-25 (Jan.); 79-84 (Feb.); 129-137 (March) 1913.

McKenzie, W.: A Case of Osteomyelitis of the Frontal Bone, Treated by Penicillin, J. Laryng. & Otol. 62:33-35 (Jan.) 1948.

Morrison, L. F.: Osteomyelitis of the Frontal Bone, Ann. Otol. Rhin. & Laryng. 64:119-127 (March) 1955.

Mosher, H. P.: Osteomyelitis of the Skull: Collective Review, Internat. Abstr. Surg. 69:417-421, 1939; in Surg. Gynec. & Obst., (Nov.) 1939.

Osteomyelitis of the Frontal Bone, J. A. M. A. 107:942-947 (Sept. 19) 1936.

——— and Judd, D. K.: An Analysis of 7 Cases of Osteomyelitis of the Frontal Bone Complicating Frontal Sinusitis, Laryngoscope 43:153-212 (March) 1933.

Putney, F. J.: Uses of Penicillin in Diseases of the Nose and Throat, J. A. M. A. 126:620-622 (Nov. 4) 1944.

Rojskjaer, C. E.: Osteomyelitis Frontalis Diffusa, with Special Attention to Its Relation to Sinusitis Frontalis: A Survey of 3 New Cases, Acta Otolaryng. 34:445-478 (Sept.-Oct.) 1946.

Salinger, S.: The Paranasal Sinuses: Review of the Literature for 1942, Arch. Otolaryng. 38:270-294 (Sept.); 373-396 (Oct.) 1943.

The Paranasal Sinuses, Arch. Otolaryng. 47: 72-102 (Jan.); 189-225 (Feb.) 1948.

Sitsen, A. E.: Über die Osteomyelitis der Schädelknochen, Monatsschr. Ohrenh. 72:729-776, 1948.

Wilensky, A. O.: Osteomyelitis: Its Pathogenesis, Symptomatology and Treatment, New York, The Macmillan Company, 1934, pp. 275-345.

Association of Osteomyelitis of the Skull and Nasal Accessory Sinus Disease, Arch. Otolaryng. 15:805-833 (June) 1932.

Williams, H. L.: Chemotherapy in Otolaryngology, Wisconsin M. J. 44:507-511 (May) 1945.

——— and Nichols, D. R.: Spreading Osteomyelitis of the Frontal Bone Treated with Penicillin, Proc. Staff Meet. Mayo Clin. 18:467-469 (Dec. 1) 1943.

Woodward, F. D.: Osteomyelitis of the Skull, J. A. M. A. 95:927-930 (Sept. 22) 1930.

The Diagnosis and Treatment of Ménière's Disease

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Prosper Ménière [1] in 1861 described a disease characterized by vertigo, tinnitus, and nerve deafness which now bears his name. He emphasized the perceptive hearing loss and pointed to the inner ear as the site of the disease. He felt that patients with this disease could readily be differentiated from those who had attacks of vertigo, nausea, and vomiting with no hearing loss. It might be added parenthetically that this is true today and that Ménière's name should not be attached to conditions other than that which he described so well. The terms "pseudo-Ménière's disease," "Ménière's syndrome complex," etc., have no place in modern medical terminology.

It was not until 1938 that the histopathologic changes in the inner ear of Ménierè's disease were described by Hallpike.[2] Hallpike obtained the temporal bones from two patients suffering from Ménière's disease who had died after intracranial section of the eighth nerve. In both he found gross dilatation of the cochlear duct secondarily involving a saccule and the utricle. Lindsay [3] and others * corroborated his findings.

Diagnosis

Ménière's disease is characterized by vertigo, tinnitus, and nerve deafness. Each of these symptoms has a peculiarity which can be discovered in a carefully taken history.

Vertigo

It is important at the outset to determine when the patient says he is "dizzy" whether he is suffering from true aural vertigo or whether he means a sensation of "blacking out" or fainting such as accompanies temporary cerebral anoxia. The common story of the first attack is that the patient was awakened from sleep by a feeling of spinning or that "the room was going round." When he attempts to move he becomes nauseated and vomits. He is conscious and can describe his sensations. Lying perfectly still in bed he is reasonably comfortable, but any movement precipitates dizziness, nausea, and vomiting. The attack may last for a few minutes or for hours or even for one or two days. At other times, the first attack of vertigo is very severe and may cause the patient to fall and to vomit. He cannot move and lies with arms outstretched grasping the ground until the attack is over or help comes. Between acute vertiginous attacks the patient is constantly unsteady, particularly on forward motion, and may say that he is apt to bump into objects and on occasions has the sensation of the horizon rising and falling as it does on a pitching ship at sea. As the disease progresses the duration and severity of the vertiginous attacks may vary considerably.

Tinnitus

The patient with Ménière's disease complains of an annoying, constant full feeling in the affected ear and of a roaring tinnitus. The sound is described as that heard on listening to a conch shell. It may be described as the sound of a wind in the forest or the sound heard near a telephone pole when the wind is blowing. Some patients describe the sound as that of steam escaping but the pitch is low. Patients also describe intermittent tinnitus of a high pitch (whistles, bells, etc.), but these sounds are

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* References 4-6.

not particularly annoying. It is the low-pitched constant roaring that is very bothersome and makes it difficult for the patient to hear even if one ear is normal.

Perceptive Deafness

Although the patient's chief complaint is of vertigo, he offers the information that he has difficulty in hearing. The usual complaint is of fullness in the affected ear, usually becoming worse just before an attack of vertigo. The patient notices a great fluctuation in his ability to hear and says that sometimes he can "hear perfectly" and at others is "quite deaf." Careful inquiry usually elicits the fact that some hearing loss preceded the onset of vertiginous attacks. The audiometric findings are usually quite characteristic.

Pure-Tone Audiogram

In the early stages of the disease the pure-tone audiogram shows a greater loss in the low tones than in the high. As the disease progresses the audiometric curve tends to flatten and the loss may be equal throughout the scale. Later in the disease there is usually increasing loss in the high tones.

Speech Audiogram

Characteristically there is a much greater discrimination loss for speech than would be suspected from the pure-tone audiogram. While the threshold loss for speech may be no greater than 40 db., the discrimination score is usually less than 50% and more often is below 30%.

In 127 cases recently reviewed, 94 (74%) had discrimination scores of less than 30%, 27 (21%) had discrimination scores of between 30%-50%, and only 6 (5%) had better than 50% discrimination.

Recruitment is always present in the affected ear, and the patient complains that loud sounds are very annoying. In unilateral cases there may often be evidence of hyperrecruitment.

Diplacusis

In most instances the patient notices a very definite difference in pitch in the two ears when tested with tuning forks. Some-

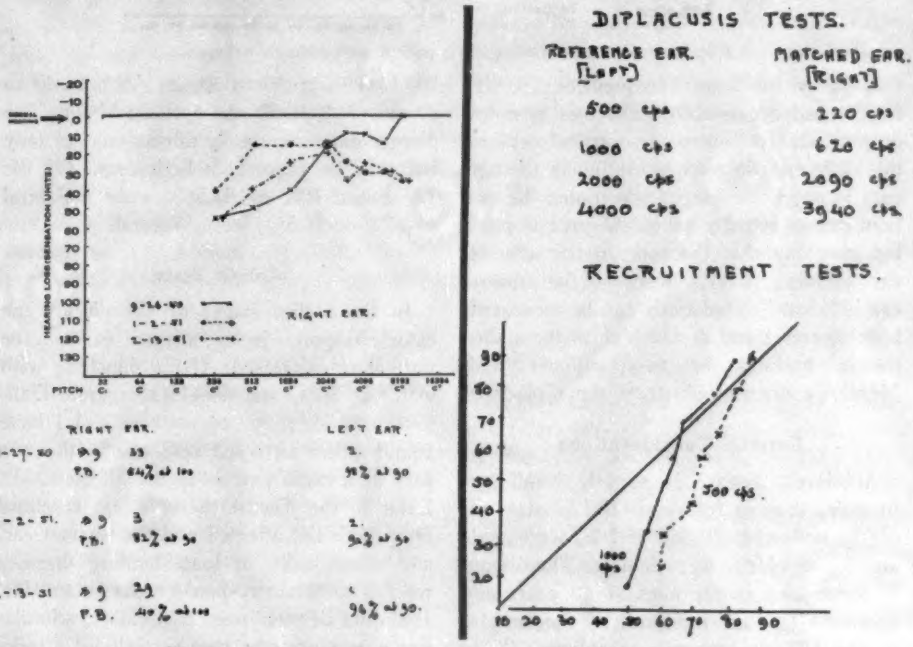

Fig. 1.—Audiometric findings in a patient with Ménière's disease.

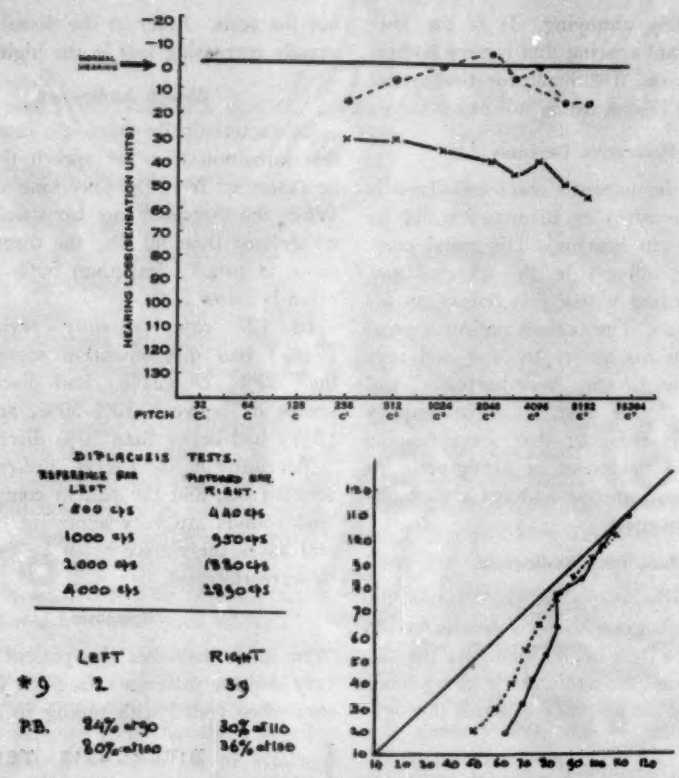

Fig. 2.—Audiometric findings in a patient with Ménière's disease.

times only the lower frequencies are affected, and occasionally diplacusis is noted only at one frequency. In musical persons this difference in pitch sensation in the two ears is very annoying. Sometimes the patient cannot actually tell a difference in pitch but may say that the tone in the affected ear sounds "fuzzy," whereas the normal ear is "clear." Diplacusis can be measured, and Figures 1 and 2, which show the audiometric findings in two patients with Ménière's disease, illustrate the diplacusis.

General Considerations

Ménière's disease is slightly commoner in males than in females. Of 127 cases recently reviewed, 70, or 55.1%, were male and 57, or 44.9%, were female. The disease is commonest in the ages of 40 years and upward. The age incidence by the decades in our 127 patients was as follows: 20 to 29, 11 (9%); 30 to 39, 26 (21%); 40 to 49, 46 (36%); 50 and up, 44 (34%). The disease more commonly affects one ear only but may be present in both ears. Of the 127 cases, 106, or 83.5%, were unilateral and 21, or 16.5%, were bilateral.

Caloric Tests

In the earlier stages of the disease the caloric response in the affected ear may be normal or depressed. Using douching with water at 30 C and 44 C (Fitzgerald-Hallpike method [7]), my co-workers and I have found either an equal response in the two ears or a canal paresis in the affected side. Later in the disease there is less response from both the affected and the normal ear, and occasionally in long-standing diseases we fail to obtain response from either side. It should be mentioned that caloric stimulation reproduces the true vertiginous attacks

of Ménière's disease. Those patients who may complain of "dizziness" which is not true vertigo can easily indicate the difference between the sensation produced by caloric stimulation and their dizzy spells. Spontaneous nystagmus is absent unless the patient is seen during an attack, and then it is present and may be toward either the normal or the affected ear.

Although Ménière's disease may be difficult to differentiate from many central nervous system lesions, it is probably most easily confused with early lesions of the cerebellopontine angle. The hearing in cerebellopontine-angle lesions becomes steadily worse and does not have remissions, there is absence of complete recruitment, and the discrimination for speech is usually relatively good as compared with the threshold loss. Caloric tests usually show a markedly diminished response on the affected side, and commonly there is a spontaneous nystagmus present.

Medical Treatment

Because of the normal remissions of the disease, it is very difficult to assess the value of any therapeutic procedure. The objectives of therapy are to eliminate the vertiginous attacks or, at least, to diminish their intensity and frequency and, if possible, to reverse the hearing loss. In the light of the spontaneous remissions of symptoms, an estimate of the efficacy of any therapy becomes a matter of clinical judgment and of observation over a considerable period of time.

Williams [8] has emphasized that there is much clinical evidence to support the theory that Ménière's disease is the result of vasomotor labyrinthine ischemia. There are many factors to be considered in the etiology of vasomotor ischemia, whether of the labyrinth or elsewhere in the body. I † discussed these in relation to vasomotor rhinitis and pointed out that allergy (hypersensitivity) and antigen-antibody reactions, endocrine dysfunction, and psychosomatic factors all play a part, severally or in combination. I believe this is equally true of Ménière's disease as of vasomotor rhinitis. The importance therefore of a careful history and general physical examination is obvious. Accepting the theory that vasomotor labyrinthine ischemia is due to vasospasm of the cochlear and vestibular vessels, vasodilator drugs have been used in an attempt to control the vertiginous attacks and to improve the hearing. Nicotinic acid in doses sufficient to cause flushing is given three or four times daily. This may be combined with methantheline (Banthine) or propantheline (Pro-Banthine). Williams [8] recommends methantheline, 100 mg. every six hours as a therapeutic dose and 50 mg. every six hours as a maintenance dose, combined with vasodilators and has seen cases in which the hearing has been markedly improved on this regimen. He says that vertiginous attacks are usually relieved. In my experience there has been amelioration of the vertiginous attacks, but I have not seen improvement in hearing with this regimen.

With the knowledge of the remarkable effect of 2-acetylamino-1,3,4-thiadiazole-5-sulfonamide (Diamox) in glaucoma and in the hope that the secretion of endolymph might be inhibited as in the secretion of aqueous matter from the ciliary body, Diamox was used clinically in a series of patients with Ménière's disease. In two patients there seemed to be a direct relation between the administration of Diamox and the lessening of the vertiginous attacks, but in none was there any appreciable change in the hearing.

In 1954 Derlacki [11] reported good results in the treatment of Ménière's disease from the use of small doses of histamine. (He uses the "optimum dosage technique" and begins with the subcutaneous injection of 0.1 cc. of 1:100,000,000 dilution of histamine diphosphate. If no noted effect follows, 0.1 cc. of 1:10,000,000 dilution is given one to three days later. The dosage is thus increased until the amount

† References 9 and 10.

which will just relieve the symptoms is reached. Once this dose is found it is increased until that amount is reached which will relieve the symptoms for three to seven days. He used this therapy in conjunction with a salt-free diet.) Although it is difficult for me to understand how such minute doses can be effective either as a vasodilator or in producing a possible desensitizing effect, I tried the method.

My routine is to do an intradermal test with dilutions of 1:1 hundred million, 1:10 million, 1:1 million, and 1:1 hundred thousand and start with 0.1 cc. of the dilution that gives the least positive reaction. The dose is increased by 0.1 cc. each third or fourth day until relief is obtained. It is interesting that I have seen cases in which the vertigo and tinnitus have been relieved and the hearing greatly improved with this therapy. Of further interest is the fact that in two patients, when the dose was increased to what apparently was in excess of the optimum, the hearing was again made worse and the tinnitus and dizziness recurred.

Case 1.—A 39-year-old man was first seen Dec. 1, 1955, with a history of loss of hearing and tinnitus for one and a half years. The hearing loss was also accompanied by mild dizzy spells, which were insignificant until three months prior to his examination. At that time he noticed severe vertigo, accompanied by increased tinnitus in the right ear, with falling, nausea, and vomiting. He stated that his hearing varied considerably, sometimes being excellent and other times very bad. He had also noticed some variation in the hearing of the left ear.

Examination of the ears, nose, and throat was essentially normal. Fork tests demonstrated a diplacusis at 512 and 1024 frequencies. There was complete recruitment in the right ear. Table 1 shows the pure-tone thresholds and hearing tests at various dates.

The patient was titrated to histamine and showed a positive reaction to 1:1,000,000 dilution. He was treated with histamine in increasing doses, and it was remarkable that when the dose was increased to 0.5 cc. his hearing diminished. When the dose was properly adjusted at an optimum of 0.3 cc., the hearing reversed and has remained good. Vertigo and tinnitus are completely relieved.

Case 2.—A 57-year-old man, first seen in October, 1954, came in giving a history of having had a tinnitus in the right ear some three or four years prior to being seen. This was treated by an otolaryngologist, with relief. Subsequently the left ear had the sensation of fullness and a marked tinnitus occurred in the left side. He found it uncomfortable to have two people talking at the same time, the radio was unpleasant to listen to, and with the onset of the fullness and tinnitus in the left ear he had dizzy spells. One month prior to being seen he had had a severe dizzy spell while driving his car. He was nauseated, but the attack only lasted about an hour. Subsequently a few days later he had another spell. He had been seen in another hospital prior to admission here, at which time a complete neurological examination was found to be negative.

On examination the ears, nose, and throat were essentially normal. He had a marked diplacusis with the 512 and 1024 frequencies. Hearing tests were performed and are illustrated in Table 2.

It was interesting that he showed a positive reaction to histamine of 1:1,000,000 dilution and that when he was receiving 0.3 cc. the hearing in the left ear was improved so that, at least for speech, the hearing was normal. When the dose was increased the hearing again was made very bad in the left ear; his tinnitus and dizziness recurred. On proper adjustment of the dose of histamine, again marked improvement in hearing occurred, particularly as noted by the discrimination score. He was continued on optimum doses of histamine, with a maintenance of hearing in the left ear at normal for speech, until June, 1955. The intervals between injections were now lengthened, and he had an injection once every two weeks. In July, 1955, he had one severe dizzy spell and the hearing decreased. He was then started on injection once a week during July, and on the 28th of July all injections were stopped and he was given 1000γ of cyanocobalamin (Vitamin B_{12}), to be taken by injection once a day; he was next seen in September. In September it was noted that the hearing in the left ear had then regressed to a 30 db. loss for speech with 64% discrimination, and although histamine has been instituted again at regular weekly injections, the hearing has been maintained at a level below that which was optimum.

In spite of the work of Mygind and Dederding [12] and of Furstenberg, Lashmet, and Lathrop,[13] I have been unimpressed with the value of a salt-free diet in the treatment of Ménière's disease, especially in the light of Perlman's [14] recent report.

In recent years cyanocobalamin has been found to exert a remarkable effect in various neuropathies. Fields and Hoff [15] found relief of pain in trigeminal neuralgia by the use of massive doses of cyanocobala-

TABLE 1.—*Results of Hearing Tests in Case 1*

	Right								Left								Speech			
																	Right		Left	
Date	256	512	1024	2048	2896	4096	5792	8192	256	512	1024	2048	2896	4096	5792	8192	T.L.	Dis.	T.L.	Dis.
12/1/55	45	55	65	50	50	60	/	/	25	30	15	10	45	50	65	60	85	6%	16	80%
12/29/55	40	50	60	35	50	65	/	/	30	40	40	25	50	55	60	65				
1/ 9/56	30	35	35	30	55	65	/	/	45	40	45	20	50	55	65	60	27	32%	29	54%
2/ 6/56	25	30	25	15	40	65	/	/	30	45	50	30	50	55	65	60	15	64%	43	24%
2/20/56																	16	72%	30	50%
3/ 5/56	25	30	20	15	45	65	/	/	20	5	15	20	50	55	65	60	12	66%	0	80%

The meaning of the abbreviations is as follows: T.L., threshold loss; Dis., discrimination.

TABLE 2.—*Results of Hearing Tests in Case 2*

		Left Ear								Speech	
Date	D.H.	256	512	1024	2048	2896	4096	5792	8192	T.L.	Dis.
10/18/54		35	35	40	25	25	30	35	35	46	30%
10/21/54	0.1										
10/25/54	0.2										
10/28/54	0.3	25	20	20	15	25	35	20	15		
11/ 1/54	0.5									2	82%
11/ 8/54	1.0										
11/11/54	0.4									30	54%
11/15/54	0.5	65	60	60	35	35	35	35	25		
11/18/54	0.4										
11/22/54	0.3										
11/26/54	0.2										
11/29/54	0.2	40	35	35	35	40	45	35	35		
12/ 2/54	0.15	25	25	25	35	30	35	35	30		
12/ 6/54	0.2										
12/ 9/54	0.15	30	25	15	15	25	40	25	25	11	82%
12/23/54	0.2	45	40	25	20	25	30	25	25		
12/30/54	0.2	45	45	25	15	20	20	25	20	17	78%
2/11/55										14	84%
3/31/55	0.3 0.4	25	15	15	20	25	30	35	25	2	80%
6/ 9/55	1x wk.									6	84%
9/15/55	none June-Aug.	50	45	35	30	35	45	40	35	30	64%
3/ 8/56	0.3 1x wk.	50	55	50	35	40	45	50	45	18	52%

The meaning of the abbreviations is as follows: D.H., doses histamine; T.L., threshold loss; Dis., discrimination.

min. With the prospect of possibly preventing deterioration of the eighth nerve and in the hope of improving the hearing, some patients were treated with large doses of cyanocobalamin. The two following case reports are interesting in this regard.

CASE 3.—A 27-year-old white man, first seen in October, 1951, was referred with a diagnosis of Ménière's disease, which had been made by a competent otolaryngologist sometime before. The history went back to 1943 and started with tinnitus; dizzy spells started in 1949. These had increased in severity and frequency. The attacks had lasted four or five hours and were accompanied by nausea and vomiting. The patient had been treated with nicotinic acid, dimenhydrinate (Dramamine), and histamine before being seen by me and was referred to have destruction of the labyrinth because the dizzy spells were interfering with his work and were of real danger to him.

Examination in October, 1951, revealed typical findings of Ménière's disease affecting the right ear. The pure-tone audiogram showed a flat loss throughout the scale, with an average of 45 db. Speech tests revealed a threshold loss of 39 db. with 44% discrimination, and the left ear was normal. The right ear showed hyperrecruitment. Caloric tests showed a diminished function on the right side.

The patient was anxious to postpone surgery until after Christmas and was therefore advised to continue with the medication of histamine, dimenhydrinate, and nicotinic acid and return in January, 1952.

He came on Feb. 7, 1952, complaining that he was still off balance and that loud noises increased the feeling of dizziness. The tinnitus had continued, although it had been somewhat less marked for two days before he was seen. Hearing tests repeated at this time showed the hearing in the right ear to be nearly normal. Speech threshold loss in the right ear was 16 db.; the discrimination score was 94%. Caloric tests still showed diminution of the function in the right side. The patient was started on cyanocobalamin, 1000γ intramuscular injection, plus 500 mg. of ascorbic acid once a day, the dose to be used for 30 days.

He was seen again in March, 1952, and remarked that he had felt better in the last 30 days than he had for many years. He had no tinnitus; this had completely disappeared for about two weeks. He stated that when he first started taking the cyanocobalamin he had had dizzy spells for the first 10 days but since that time the dizziness had been completely relieved. Caloric tests showed equal function in both labyrinths at this time. Hearing tests continued to be normal. He was given a maintenance dose, 1000γ of cyanocobalamin, once a week and was seen during 1952, at which time he had no dizzy spells. In the latter part of September, 1952, he was told to discontinue all medication and notify us if there was any return of symptoms. He came back in October, 1952, having had a severe dizzy spell, and was again seen, in November, 1952, when he had a very severe dizzy spell the week before we saw him. At that time we gave him a placebo in place of the cyanocobalamin. It was put up in ampules exactly the same as those of the cyanocobalamin and exactly resembled the cyanocobalamin. He was not informed of any change in medication. He returned in January, 1953, saying that he had had dizzy spells even while taking the material he had been given and vouchsafed the information that he felt there was something wrong with the last batch of medication because it had not relieved his symptoms. He was again started on cyanocobalamin, in January, 1953, and at that time was given a sufficient amount for 30 daily injections. In February, 1953, he communicated with us, saying he was very well, had had no dizziness, and his hearing was normal. There was no tinnitus. He had been put on a maintenance dose of cyanocobalamin and took it during 1953 and 1954.

In January, 1955, I took him off all medication and asked him to report from time to time. He was seen again, in May, and has been under the care of his otologist since that time, having taken no further medication and having had no further attacks of dizziness. Hearing is still normal.

CASE 4.—A 74-year-old white man was seen in the Ear, Nose, and Throat Clinic in February, 1953, with a complaint of a roaring tinnitus in the left ear which had started two and a half months prior to his being examined, together with a hearing loss which was quite marked in the left ear and attacks of dizziness which appeared to be true aural vertigo. A preliminary diagnosis was made of senile degeneration of the eighth nerve with a possibility of tubotympanitis on the left side. Inflations of the Eustachian tubes were done, with no remarkable change.

The hearing studies were done and are illustrated in Table 3. There was a definite recruitment in the left ear, a diplacusis, and an intolerance to loud sounds. Caloric tests revealed a hypoactive left labyrinth.

The diagnosis was made of Ménière's disease on the left side, and he was started on medication with β-pyridyl carbinol (Roniacol) and thiamine chloride. On this medication he felt better; he was seen in June, 1953, still complaining of tinnitus and dizzy spells, but these were not as severe as before.

In July of 1953 and, again, in August and September he was seen in the same condition. In September, 1953, he was taken off all medication

TABLE 3.—*Results of Hearing Tests in Case 4*

	Right								Left								Speech			
																	Right		Left	
Date	256	512	1024	2048	2896	4096	5792	8192	256	512	1024	2048	2896	4096	5792	8192	T.L.	Dis.	T.L.	Dis.
2/19/53	0	0	0	15	30	35	25	25	25	20	10	45	50	40	65	45				
3/25/53	0	0	0	30	45	50	50	55	50	35	45	35	45	50	50	45				
5/ 5/53	0	0	0	45	55	55	55	60	45	35	30	25	45	45	55	35	11	66%	42	28%
2/ 9/54	0	0	−5	40	55	55	60	60	20	25	10	15	40	45	55	40	5	60%	16	76%
2/ 1/55	−5	−5	0	35	50	55	50	55	30	35	35	10	45	45	55	40	8	68%	28	72%
10/18/55																	6	70%	30	54%
12/13/55																	6	64%	31	56%
2/ 7/56																	3	70%	27	60%

The meaning of the abbreviations is as follows: T.L., threshold loss; Dis., discrimination.

and given cyanocobalamin, 1000γ daily, by intramuscular injection. He returned in October and November and said that his tinnitus was very much improved, his hearing seemed to be better, and that he had had no unsteadiness. On retest of his hearing in December, 1953, there was found a remarkable improvement in the left side. He was put onto a maintenance dose of 1000 γ cyanocobalamin once a week and was kept on this during 1954, and in July, 1955, he was given cyanocobalamin, to be taken by mouth, the dose being 1000 γ three times a week.

He returned in October, at which time his hearing was tested, revealing a considerable diminution of hearing in the left ear. The dose of cyanocobalamin was then increased to 1000 γ by mouth once a day, and he was seen in December, when, again, there was some improvement in the discrimination score. The dose, 1000 γ, was then increased to three times a day. When the patient was seen in February, 1956, the speech threshold showed a remarkable improvement.

These examples of different therapeutic measures serve only to emphasize that no one method can be said to be the treatment of choice in Ménière's disease. The objective of treatment is to overcome the spastic atonic state of the cochlear vessels and to relieve the vasomotor labyrinthine ischemia. Essential is the investigation of the possible etiologic factors, such as allergies, endocrine dysfunctions, psychosomatic factors, etc., and treatment demands a combination of therapeutic approaches which will differ in each case. There is no one specific medical therapy for Ménière's disease.

Surgical

In long-standing cases of Ménière's disease when one ear only is affected the patient's hearing is such that it is not only useless to him but may, because of the tinnitus and diplacusis, be a real detriment. Further, the vertiginous attacks, if unrelieved by medical therapy, may be severe enough to make work impossible and life intolerable. In such instances surgical destruction of the ear is easily accomplished.

Relief of the vertigo by intracranial section of the eighth nerve is only mentioned to be deprecated. Differential section was proposed by Dandy[16] and performed in many instances. The objective of preserving the hearing by differential section was ill-conceived when it is realized that the hearing, with a discrimination loss such as it usually is, is useless. The only valid reason for differential section is in the case of bilateral hearing loss when one ear can be blamed for the vertiginous attacks.

In 1943 Day[17] suggested coagulation of the labyrinth as a means of relieving vertiginous attacks. Using a postauricular incision he exposed the horizontal semicircular canal, and having made an opening into the anterior end of the canal he inserted an electrode and coagulated the vestibular contents. He was able on many occasions to preserve the hearing but found in most instances that the patient later returned to have the hearing destroyed, to be rid of the diplacusis and in the hope of relieving the tinnitus.

Cawthorne[18] advocates labyrinthotomy and removal of the membranous labyrinth with fine forceps.

I believe that for surgical intervention to be successful complete destruction of the vestibular labyrinth is essential. In five cases in which I performed coagulation as described by Day, I had later to reoperate. The patients, although they had no vertiginous attacks, were unsteady, particularly on forward motion. In order adequately to destroy the labyrinth I find it best to remove the incus and make a large opening into the vestibule, combining the anterior ends of the superior and horizontal canals and the vestibule into one large opening. The membranous labyrinth is then extracted and great care exercised to be certain that all neural elements in the vestibule are destroyed. The opening into the vestibule is then packed with bone, saved from the exenteration of the mastoid cells, and the incision is firmly closed. When adequate exenteration of the soft tissues in the vestibule has been accomplished, the result is a completely dead ear, with no labyrinthine function and no further vertiginous attacks. Of 37 cases operated upon for Ménière's disease, 35 were completely relieved of

their symptoms, while 2 developed hydrops in the second ear.

Summary

Ménière's disease is characterized by vertigo, tinnitus, and perceptive deafness. It is subject to spontaneous remissions.

There is clinical evidence to support the theory that Ménière's disease is the result of vasomotor labyrinthine ischemia. Therapeutic efforts demand a careful history and physical examination to determine, if possible, the factors—allergic, endocrine, and psychosomatic—which may have etiologic significance.

No one medical therapy is effective in all cases.

Surgical destruction of the labyrinth gives relief in those cases in which one ear only is affected and should be considered after all medical management has failed.

REFERENCES

1. Ménière, P.: Gaz. méd. Paris 16:239, 379, and 597, 1861.

2. Hallpike, C. S., and Cairns, H.: J. Laryng. & Otol. 53:625, 1938.

3. Lindsay, J. R.: Arch. Otolaryng. 35:853, 1942.

4. Hallpike, C. S., and Wright, A. J.: J. Laryng. & Otol. 55:59, 1940.

5. Altmann, F., and Fowler, E. P., Jr.: Ann. Otol. Rhin. & Laryng. 52:52, 1943.

6. Lindsay, J. R.: Laryngoscope 56:325, 1946.

7. Fitzgerald, G., and Hallpike, C. S.: Brain 65:115, 1942.

8. Williams, H. L.: Ménière's Disease, Springfield, Ill., Charles C Thomas, Publisher, 1952.

9. Walsh, T. E.: Laryngoscope 60:360, 1950.

10. Walsh, T. E.: Ann. Otol. Rhin. & Laryng. 64:466, 1955.

11. Derlacki, E. L.: Laryngoscope 64:271, 1954.

12. Mygind, S. H., and Dederding, D.: Acta oto-laryng. 17:424, 1932.

13. Furstenberg, A. C.; Lashmet, F. H., and Lathrop, F.: Ann. Otol. Rhin. & Laryng. 43:1035, 1934.

14. Perlman, H. P.; Goldinger, J. M., and Cales, J. O.: Laryngoscope 63:640, 1953.

15. Fields, W. S., and Hoff, H. E.: Neurology 2:131, 1952.

16. Dandy, W. E.: Arch. Surg. 16:1127, 1928.

17. Day, K. M.: Laryngoscope 53:617, 1943.

18. Cawthorne, T. E.: J. Laryng. & Otol. 58:363, 1943.

Objective Audible Clicking in Ear

Presentation

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Tinnitus aurium is a clinical entity that is quite prevalent. A simple definition of tinnitus is noise in the head. It may be transitory or constant, rhythmic or irregular, soft, medium loud, or loud. It may or may not be synchronous with the pulse rate. It may be totally involuntary or it may be partly or wholly controlled under certain conditions.

The noise may be a hum, a click, a tick, or soft blowing in character. Usually it is audible only to the patient, in which case it is termed subjective tinnitus. However, in rare instances, the head noise is audible to the examiner as well. In this case, it is described as objective.

Certain cephalic murmurs, caused by an arterial aneurysm or an engorged cranial vessel, can be heard by means of the stethoscope. This type of cephalic auscultation was formerly practiced extensively, but now it is rarely resorted to. Cephalic murmurs differ from cardiac murmurs in that they are first heard and discovered by the patient himself. They are also called pulsatory. They differ considerably in pitch and intensity from the extrinsic objective head noises that do not originate in a blood vessel. Graphic recordings have been made of these intrinsic vascular head noises, especially by means of the Mannheimer phonocardiograph.

However, the situation is different in cases of objective tinnitus where there is no relationship between the head noise and the pulse beat. Tinnitus may or may not be accompanied by other complaints. There may be associated clinical findings that have a direct bearing on the head noise. However, often the patient is mainly concerned with the tinnitus from which he is seeking relief.

There may be no accompanying loss of hearing or no demonstrable ear pathology. However, if the audible noise is loud enough, it may have a masking or diverting effect on the hearing in either or both ears.

The etiology of objective tinnitus is not definitely known. Aside from the hum or bruit or murmur associated with cranial vascular anomalies, the true etiology can seldom be determined.

Nonvascular, or vibratory objective, tinnitus is usually heard as a clicking or ticking noise and is apparently caused by muscular contractions involving the levator palati muscle, the Eustachian muscle fibers, the stapedius, or the tensor tympani muscle.

These cases are rather rare, but on close examination one may obtain a history of an associated facial nerve paralysis at their onset. At times, there may be present vertical clonic contractions of the soft palate, synchronous with the auditory click. Some observers have designated this type of head noise as pseudotinnitus, because they ascribe the sound as being conducted from the soft palatal muscle to the ear via the Eustachian tube, or by the way of contiguous structures. The following case came to my attention, Oct. 28, 1953.

The patient was a man 31 years old, whose chief and only complaint was a ticking or clicking noise

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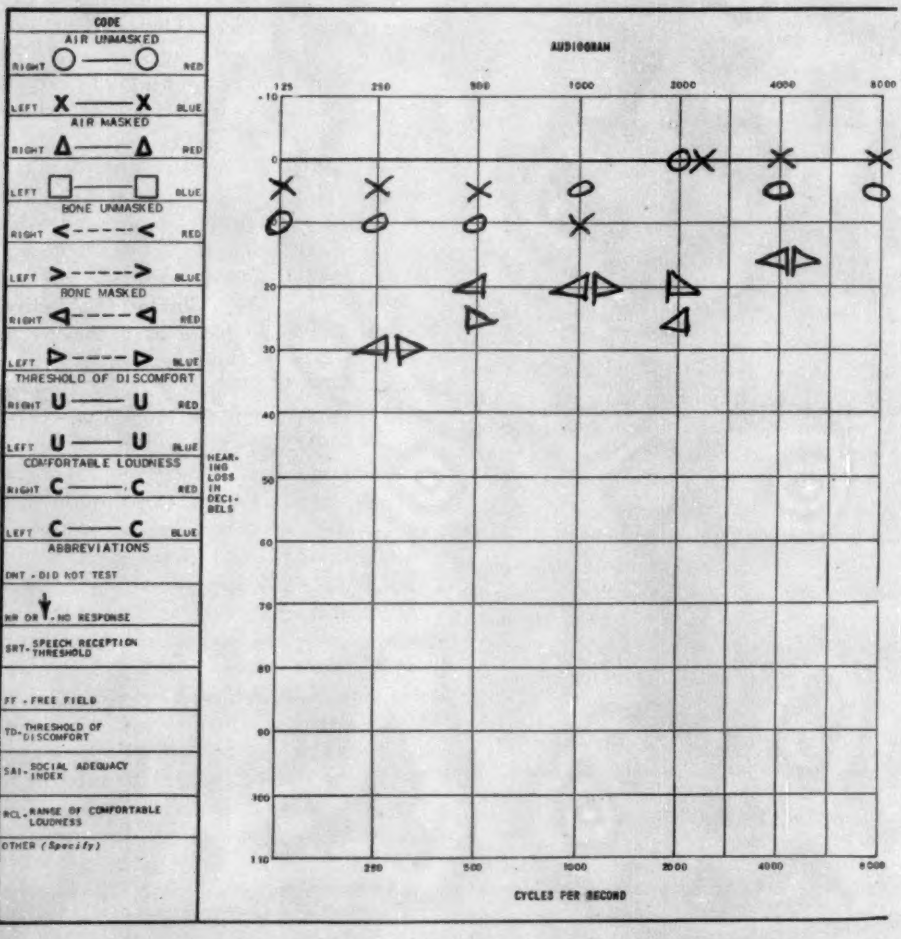
in his right ear. This noise was constant and dated back to 1944, when he developed a right-sided Bell's paralysis. The clicking sound became apparent soon thereafter, but he did not realize, until a year and a half later, that it could also be heard by anyone who was located within 1 or 2 ft. distant from his ear. Actually, his fiancée one day asked him to remove the watch from his coat pocket, while they were sitting near each other on the sofa. He then realized that his head noise could be heard by others than himself. Examination revealed no abnormalities in his ears, tympani, nose, throat, or sinuses. His hearing was normal bilateral, the conversation tone in a semiquiet room being 20 ft. in each ear. His Rinne test was positive bilateral; Weber lateralized to the right; Schwabach and air, equal and normal in each ear. There were no obvious residuals of right-sided facial palsy. A clicking, irregular sound was heard by the examiner, coming from the patient's right ear, at a distance of 18 in. This clicking noise was also heard rather faintly coming from his other ear, if the examiner placed his ear in close contact with the patient's left ear. This was, undoubtedly, a relayed echo coming from the involved ear. This sound was not synchronous with his pulse beat. It occurred from 116 to 150 times a minute and was arrhythmic. When he inhaled, it decreased in frequency, and when he exhaled it became accelerated. The sound resembled that produced by clicking two fingernails together. He could stop the tinnitus voluntarily by holding his breath or by breathing rapidly and shallowly. No definite motility of his tympanum was noted when the Stieglitz or Valsalva tests were performed. Air and bone, pure tone audiometry revealed normal responses, as illustrated by the accompanying audiogram.

There was no nystagmus, and no demonstrable



evidence of nerve degeneration on either side of his face. There was good closure of the eyelids, good bilateral forehead wrinkling, no facial droop, no tongue deviation, and no evidence of anosmia or ageusia. Visualizing the Eustachian tube through the nasopharyngoscope and inflating the tube produced cessation of the clicking sounds. There was also observed arrhythmic vertical jerking or clonic movements of his soft palate. These movements were practically synchronous with auditory click and showed the same variations in frequency as that of the latter. These movements can actually be likened to a vertical nystagmus as seen in the eyes.

A diagnosis was made of objective audible tinnitus with a nonactive otitis media catarrhalis chronica, right ear. Subjectively, the patient stated that he experienced a feeling of tightness on the right side of his face extending from the outer canthus, across the temperoparietal region to about 1 in. postauricularly, the lower facial level being below the zygomatic and malar region.

Incidentally, his tinnitus could be heard by stethoscope over this area as well. X-rays of his mastoids, temperomandibular joints, internal auditory meati, and sinuses revealed no abnormalities. Laboratory tests showed normal blood counts. The CO_3 volume % was 22 mEq (normal being 23 to 27 mEq per 100 cc.); calcium was 9.7 mg. per 100 cc. (normal is 9.5 to 11.5); inorganic phosphorus was 3.5 mg. (normal); electrolytes—potassium was 4.2 mEq per liter (normal 3.8 to 5.3 mEq per liter); sodium was 143 mEq per liter (normal is 136 to 145 mEq per liter).

It might be added here that his history revealed presence of residuals of right-side facial nerve degeneration reaction as late as 1950. At that time there were also noted a mild atrophy of the right cheek and a slight droop of the right side of the mouth. These findings can be considered as mild residuals of an old degenerative reaction of the seventh nerve. He was seen by me again in December, 1955, and most recently on Jan. 11, 1956. At none of these examinations were there different clinical findings than those previously noted. At present, tests show very minimal evidence of nerve degeneration. Apparently there has been fairly complete nerve regeneration with possible admixture of other nerve fibers.

In trying to rationalize or visualize the neuroanatomical basis to explain this case, one should describe the potential nerves and muscles involved. First to be considered is Arnold's nerve. This structure comprises the sensory fibers of the vagus or 10th cranial nerve. It leaves the jugular foramen through its medial wall and joins the seventh or facial nerve about 4 mm. above the sternomastoid foramen. There it joins some facial fibers, to be known as the postauricular (sensory) nerve. It has some distribution to the ear drum and to the external auditory meatus. It joins the seventh nerve just above the sternomastoid foramen. The facial nerve branch supplying the stapedius muscle originates at a distance from Arnold's nerve. The nerve to the stapedius arises from the seventh nerve close to the geniculate ganglion. Some sensory fibers infiltrate the stapedius.

Wrisberg's nerve, or nervus intermedius, is part of the seventh nerve. It is mostly sensory, but transmits some parasympathetic and motor impulses.

Bell's nerve, or the phrenic nerve, is purely respiratory in function.

Motor nerves of the palate derive from the trifacial nerve, which controls the tensor tympani muscle, and from the facial nerve, which innervates the stapedius muscle. A question that arises is "Do parasympathetic fibers ever regenerate?"

So far the nerve distribution has been discussed. Now let us consider the various muscles which may be directly or indirectly involved in a case of this kind.

The internal pterygoid muscle, supplied by the seventh nerve, arises from the pterygoid fossa of the sphenoid and is inserted into the inner side of the angle of the mandible.

The tensor tympani muscle arises from the cartilaginous part of the Eustachian tube, from the adjacent part of the greater wing of the sphenoid, and from the bony walls of the semicanal which encloses it. It ends in a round tendon to be attached to the manubrium of the malleus near its neck. It is supplied by the motor division of the fifth nerve through the tensor tympani branch from the otic ganglion.

The stapedius muscle arises within the eminence of the pyramid, makes its exit through an opening at its apex, and is inserted on the posterior surface of the

neck of the stapes. It is supplied by a branch of the facial nerve.

The levator veli palatini originates from two points: (1) the inferior surface of the petrous portion of the temporal bone; (2) the lower margin of the cartilage of the Eustachian tube. It is inserted into the aponeurosis of the soft palate. It is supplied by the pharyngeal branch of the vagus. Its functions are to lift the velum of the soft palate and to narrow or close the opening of the Eustachian tube.

The tensor veli palatini arises from the scaphoid fossa and angular spine of the sphenoid and from the cartilaginous wall of the Eustachian tube. It is inserted into the horizontal plate of the palate bone and into the soft palate aponeurosis. The nerve supply is the mandibular branch of the trigeminal, through the tensor palatine branch of the otic ganglion. It tightens the soft palate and opens the Eustachian tube during deglutition.

Another phenomenon that cannot be ignored is antidromic conduction, possessed by some nerve dendrites. At times these dendrites may penetrate from the skin into a blood vessel, causing dilation, or they may infiltrate into muscle tissue. In such cases they may produce an impulse in the opposite direction through the neurons. Thus, in dendritic regeneration, some sensory fibers can become intermingled with motor fibers, and this may result in permanent clonic muscle contractions. These contractions in turn may be translated into audible sounds by passing through contiguous and hard tissues.

Cochlear lesions can initiate or excite tinnitus. A muscle contraction does not consist of a heavy tug or pull, but it possesses a periodic or vibratory nature which is translated into sound via the contiguous structures through which these impulses are transmitted.

Any such abnormality in the middle ear can cause disturbance between the ossicular chain and the two middle ear muscles. In this manner, either the stapedius or the tensor tympani assumes a repetitive state of tension, thus producing the tinnitus by the vibrations communicating with the cochlea which are, in turn, translated into sound. The clicking sound accompanying palatal contractions is presumably caused by the Eustachian tube being forcibly opened by each contraction.

Search through scientific literature reveals the rarity of such cases. Most of those reported are of a vascular hemic type. In the latter cases, it is possible to institute remedial measures with more or less success.

However, in these long-standing vibratory or muscular auditory clicks or head noises, the prognosis is rather poor, as far as alleviating the condition is concerned. No attempt is made in this presentation to discuss treatment of this condition. To mention a few methods—they are nerve section, mastoid surgery, sedatives, antihistaminics and amphetamine.

Summary

A case of auditory objective clicking of the ear is described. This clicking or tinnitus is heard by others than the patient, at a distance from 1 to 2 ft. from the patient's ear.

This condition occurred in conjunction with Bell's paralysis, over 11 years ago, the latter condition leaving practically no residuals of facial muscle palsy.

Search through the literature back to 1904 indicates the rarity of similar conditions.

Finally, an attempt is made to explain the neuroanatomy, specifically of the actual muscles and nerves involved in such cases. So far as I can ascertain, this has not been done previously in the medical literature.

BIBLIOGRAPHY

Bárány, R.: Zur Ätiologie, Therapie und Theorie der objective-subjective Ohrgeräusche, Acta otolaryng. 16:311, 1931.

Bredlau, E. A.: Objective Tinnitus Aurium, Arch. Otolaryng. 28:193, 1938.

Elliott, E.: A Case of Clicking Ears, Brit. M. J. 2:652, 1946.

Engström, H., and Graf, W.: Recording of Objective Tinnitus, Acta oto-laryng. 41:228, 1952.

Fowler, E. P.: "Illusion of Loudness" of Tinnitus —Its Etiology and Treatment, Laryngoscope 52: 275, 1942.

Friedmann, C.: On Objective Tinnitus, Ztschr. Ohrenh. 46:373, 1904.

Goldflam, S., and Meyerson, A.: Über objective Ohr- u. Kopfgeräusche, Wien. med. Presse 36: 641 and 728, 1895.

Graf, W.: Cephalic Murmurs in Connection with 2 Cases of Objective Tinnitus, Nord. med. 28: 2499, 1945.

Hamburger, L. P.: Head Murmurs, Am. J. M. Sc. 181:756, 1931.

Iglauer, S.: Objective Tinnitus Aurium, Arch. Otolaryng. 18:145, 1933.

Jones, K. H., and Knudson, V. S.: Certain Aspects of Tinnitus, Particularly the Treatment, Laryngoscope 38:597, 1928.

Passow, A.: Objective Ohrgeräusche, Internat. Zentralbl. Ohrenh. 8:511, 1910.

Sataloff, J. J.: Objective Tinnitus: Report of a Case, Ann. Otol. Rhin. & Laryng. 58:1082, 1949.

Schubert, K.: "Über objektives Ohrenklingen und seine Entstehung," zur Arbeit von W. Kley, Ztschr. Laryng. Rhin. Otol. 29:230, 1950.

Shanks, J.: Subjective and Objective Noises in Ear ("Buzzing"), M. Rec. 161:289, 1948.

Sharp, A.: Case of Audible Tinnitus, Proc. Roy. Soc. Med. 3:54, 1909.

Weil, A.: A Case of Objective Tinnitus Auri, Laryngoscope 14:202, 1904.

Yates, D. G.: Case of Objective Tinnitus Aurium, Ann. Otol. Rhin. & Laryng. 19:734, 1910.

Young, N.: Objective Tinnitus, Brit. M. J. 1:414, 1942.

Cerebrovascular Disease in Otolaryngology

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There are a number of otolaryngologic conditions which occur because of pathological states in the cerebrovascular system. These conditions may seem obscure until they are thought of in relation to cerebrovascular disease. For example, a middle-aged patient was referred by an internist to the department of otolaryngology for the evaluation of the symptom of hoarseness. This appeared suddenly and was associated with weakness, vertigo, and ataxia. Almost everyone is acquainted with the association of hoarseness and mediastinal conditions. This had already been excluded. Examination revealed a cord paralyzed on the left side. Because of the associated vertigo and ataxia, a brain lesion was considered a possibility, and the vestibular caloric test was applied. There was a marked reduction in the vestibular response on the left side.

The symptoms of vertigo, ataxia, hoarseness, and weakness struck a note which pointed to a possible vascular lesion in an area supplied by a single vessel. Further tests were made. A numbness on the left side of the face compared with that of the right was found, about which the patient did not complain. When the larynx was examined with a hot laryngeal mirror, the same mirror was applied to the left side of the body, arm and neck, and the patient promptly withdrew. The same hot mirror was applied to the arm on the opposite side, the right, and was tolerated by the patient. In fact, he did not realize that something hot touched his body. The entire picture now seemed clear. In spite of the absence of a Horner's syndrome, a thrombosis of the left posterior inferior cerebellar artery was diagnosed. This artery supplies that region in the medulla which harbors the vestibular nuclei, the inferior cerebellar peduncle carrying vestibular impulses from the nuclei to the cerebellum, the vagus nuclei, the descending root of the fifth nerve and the spinothalamic tract carrying pain and temperature impulses from the opposite side of the body. Here, therefore, was a patient whose most apparent symptom was hoarseness, for which he was referred to the otolaryngologist for evaluation. The thinking in terms of cerebrovascular disease led to proper tests and diagnosis.

Another patient was treated surgically for a large retrotonsillar mass on the left side, which proved to be a benign gland, although very large. However, the next day the patient developed a secondary hemorrhage. The wound was tightly packed—probably too tightly—and the bleeding was controlled. The same evening a call came from the hospital that the patient went into shock and when seen revealed a hemiplegia on the opposite side and an aphasia. A Horner's syndrome was also present. The packing was removed, and bleeding was no longer a problem. The internal carotid artery is usually thought of in terms of bleeding in surgery of the tonsil or in its surrounding area. Seldom is the possibility of compression with deleterious effect given consideration. However, there it was staring one in the face, its compression causing a sudden narrowing of the middle cerebral artery with the consequences described. In addition, another situation, rarely thought of, arises, that is, the proximity of the ganglionated cord close to the internal carotid artery in the neck, which in this case inflicted the Horner's syndrome.

Murphy,[1] quoting Alvarez, reports syncope resulting from a hypertensive carotid

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sinus reflex which caused hemiplegia and even death after external massage of the carotid artery or during a tonsillectomy.

These instances, and there are others, would seem to impel the otolaryngologist to become acquainted with some vascular situations in the head which can bring about pathologic states in the field of otolaryngology.

The vascular conditions which most commonly appear on the scene are cerebrovascular thrombosis with infarction, hypertension, arteriosclerosis, aneurysm, and vasospasm—all of which may extend their effects to otolaryngologic areas.

Cerebral thrombosis with resultant softening of the brain, infarction, comprises about two-thirds of all cerebral strokes (Murphy [2]). In 60% of instances of cerebral softening no occlusion of the vessel supplying that area can be found (Hicks and Warren [3]). These may be due to thrombosis of the cervical carotids, or a poor heart action can modify blood pressure so as to cause ischemia with softening. The causes may be arteriosclerosis, inflammatory vascular disease, or trauma to the carotids which could cause thrombosis in their tributaries such as probably occurred in the case referred to above (Pratt-Thomas and Berger [4]). There may also be some blood dyscrasias, diabetes, congenital cardiac conditions, and even emotional crises which could precipitate a central infarction (Murphy [1]). The middle cerebral artery with its vast distribution to the Sylvian fissure, island of Reil, internal capsule, and striate bodies could have varying effects, depending on the extent of involvement. Weakness, confusion, vertigo, and parasthenias may appear in areas involving small vessels.

The commonest major thrombosis not infrequently encountered by the otolaryngologist is the thrombosis of the posterior inferior cerebellar artery because of its sudden onset and its resemblance to Mèniére's disease, as in the first case cited.

Little Strokes

In a discussion of vascular thrombosis the term little strokes looms large as a factor which causes difficulties in many areas and which could be overlooked because no immediate major catastrophe appears. If the strokes appear at night, they may never become known until some major disturbance appears. Alvarez* makes a pertinent plea for all physicians to recognize the importance of little strokes.

In otolaryngology, too, these little thromboses may show their effect on areas about which the otolaryngologist is consulted. These accidents often begin with a dizzy spell and a variety of conditions are suspected, including Ménière's disease. As an instance may be cited a patient in the proper age group complaining of vertigo. An audiographic and vestibular examination revealed an almost complete loss of vestibular responses in the vertical canals to the caloric test and a marked loss in hearing in the high pitch sounds. One branch of the internal auditory artery supplies both regions in which the pathology was present. A thrombosis of this end-artery was diagnosed. Ocular pareses and nystagmus, symptoms which the otolaryngologist is called upon to evaluate, may be caused by thrombosis of the minute vessels supplying the respective areas. Cases of sudden deafness, not reversible, may be a sign of such a thrombosis. These little strokes may also be forerunners of coronary thrombosis. An instance is here recalled when a patient was hospitalized for the symptom of vertigo which subsided in a few days. The patient had hypertension and in less than a year succumbed to coronary occulsion. The symptoms by which these little strokes may be suspected are dizzy spells, changes in mental behavior, a sensation of collapse, and impaired memory. There may be syncope or blackouts, and as in the instance above the condition may be mistaken for labyrinthine pathology. There may not be present any hypertension in

* References 5 and 6.

some instances, and the brevity of the duration of the symptoms sometimes may be due to vasospasm. Where autopsies have been studied, discrete brown areas in the brain have been found to show small thromboses. Some of these patients are diagnosed as having cardiac disease and are unnecessarily restricted, to their detriment.

Arteriosclerosis and Hypertension

Arteriosclerosis and hypertension are often causes of ear symptoms such as deafness, vertigo, and tinnitus. Tortuosities of vessels in the head region can cause reduction of the blood flow to parts in the inner ear and result in these symptoms. However, all other causes should be excluded, and, when possible, the retinal vessels should be studied by an ophthalmologist. The latter will frequently confirm the presence of such pathology. In view of the many drugs now available for the treatment of hypertension, otolaryngologists should make themselves acquainted with their possibilities. This is perhaps more necessary because of the harm these drugs may do rather than because of the good they accomplish. Drugs like reserpine (Serpasil), hydralazine (Apresoline), chlorpromazine (Thorazine), hexamethonium (hexamethylene-1,6-bistrimethylammonium diiodide), meprobamate (Miltown Equanil), etc., have been employed with good results in psychoses and hypertension. However, all who use them warn against complications varying from mild effects like weakness and dizziness to severer fainting spells, shock, impairment of kidney function, and various forms of cardiac involvement. Chlorpromazine and reserpine have been recommended for tinnitus, and good results have been reported. On the other hand, a patient was referred for the relief of tinnitus with the belief that hypertension was the cause. When the patient was questioned, the information was obtained that he had been taking reserpine for a long while, yet no relief of the tinnitus was forthcoming.

Aneurysms of cerebral vessels may pose a problem of differential diagnosis because they may exert pressure on several cranial nerves in the location about the circle of Willis. The commonest branches involved are the superior cerebellar artery, the posterior inferior cerebellar artery, and the posterior cerebral, all of which are in relation with some cranial nerve or it nucleus (Murphy [7]). Dandy [8] reported aneurysms of the anterior inferior cerebellar artery which implicated the fifth nerve, causing trigeminal neuralgia and the eighth, causing a Ménière's syndrome. The circle of Willis at the base of the brain, together with its various branches, crosses in close proximity to various cranial nerves, the involvement of which is most often attributable to otolaryngologic conditions. As an example may be cited involvement of the sixth nerve in the presence of otitic infections like petrositis or the Gradenigo syndrome. Pain in the eye frequently associated with traction on the vessels in the circle of Willis adds to the confusion because these otolaryngologic conditions also present such a symptom. Paralysis of the third nerve in temporal lobe abscess is not uncommon, and when the diagnosis is in doubt the existence of an aneurysm may come to mind. These examples should convey the thought that an aneurysm at the base of the brain cannot easily be dissociated from the field of otology.

Vasospasm

In the discussion of vasospasm the autonomic system boldly comes to mind. There are a number of conditions with which the otolaryngologist is daily concerned which depend on the vascular spasm to which a number of terms have been applied and which should be considered in terms of vascular behavior. Neuralgias, headache, and vertigo are examples. The autonomic system consists of a more or less independent nervous system acting in cooperation with the general nervous system. Its duty is to maintain all involuntary functions of the body which keep life going. We

become aware of this system only when something goes wrong. Heart action, gastric function, endocrine functions, secretions, and body defenses are examples. Many of these functions are executed through the circulatory control of this system. This function is supervised by a number of nuclei in the hypothalamus, together with other ganglionic cells distributed throughout the brain stem and spinal cord which function through nerve pathways to the proper organs. Examples of this influence on the circulation are the following:

1. Regulation of body temperature
   (*a*) Hypothermia is prevented by increased heat production, heat loss by vascular constriction (in animals—hair raising), elevation of metabolic rate (increased glucose, liberation of epinephrine, and increased activity of striated muscles (shivering, voluntary movements).
   (*b*) Hyperpyrexia is prevented by vasodilation, sweating, and panting.
2. Regulation of emotional expression
   Center of emotions is situated in the hypothalamus and controlled by the cerebral cortex. Examples of the results of this regulation are numerous. They include the salivation and increased gastric secretion induced by the concentration of blood in the striated muscles caused by anticipated violence, or fright, the sweating, particularly in the palms, caused by mental agitation. Unpleasant thoughts cause lacrimation.

It is quite apparent that the circulatory system is deeply involved in carrying out these functions. When there is an aberration in any bodily aspect over which this system has the responsibility of supervising, a number of reactions take place. The cause of the disturbance may be emotional, as fear or anger, or a somatic one, as exposures to temperature changes, toxins, or an attack by pathogenic microbes, or, in some instances, individual sensitivity to foods or other substances in the enviroment, as manifested in the allergic state.

The principal symptoms in which this system plays a part and with which the otolaryngologist is chiefly concerned are vertigo, headache, and a variety of forms of so-called neuralgias.

The symptom of vertigo, in recent years, has been studied intensively. Many of the associated manifestations seem to point to a spastic condition of blood vessels in the inner ear. Williams,[9] in an exhaustive monograph, reviewed most of the available experimental and clinical literature on Ménière's disease, together with his experience with a large number of cases. He presents a convincing story that in this disease, which he believes to be a specific entity, the cause of the symptoms is a vasospasm in the arterioles in the inner ear which brings about an ischemia in the tissues supplied by the arteriole. This ischemia is the irritant which brings about the vertigo as a manifestation of the reaction of the vestibular portion of the inner ear. When the arteriole supplying the cochlea is involved, tinnitus and deafness is the reaction. With the persistence of the vasospasm there is a retrograde filling and dilatation of the venule plus increased permeability of the striae vascularis, which in turn produces a hydrops or increase of fluid in the inner ear, which produces a characteristic deafness and the vertigo. When the arteriole supplying the cochlear portion of the inner ear only is involved, the deafness appears. Thus the picture of Ménière's disease without vertigo appears. The recovery and recurrence of these attacks in susceptible individuals make up the picture of Ménière's disease. With this vascular picture in mind, treatment is instituted, based on the action of drugs which influence vascular behavior through the autonomic system. Allergic reactions and emotional reactions which also are subservient to the autonomic system are also to be considered in therapy. Fowler[10] studied vascular changes as a result of sympathetic stimulation. He noticed narrowing of arterioles on such stimulation and dilatation of the venules which follow these arterioles. He also noted clumping of red cells in the retarded circulation of the venules. This is called sludging. He cites other workers who found similar phenomena. There have been

such reports in the literature since Fowler's work. Whether we believe as Williams does that Ménière's disease is a specific entity or that it is merely one form of action producing a labyrinthosis which could be produced by stimuli other than allergy or emotional stress matters very little in this discussion. The important situation is the reaction of the vascular system in the inner ear and the fact that the cerebrovascular system holds an important position in the field of otolaryngology. It is characteristic of vasospasm here or in other parts of cerebral circulation that the effect is temporary and function will be completely restored at least in the beginning before permanent damage is developed in the affected tissues supplied by these vessels. It is also of importance that the condition responds to antispasmotic therapy.

Headache

In the comments on headache only those headaches are referred to with which patients come to the office of the physician time and time again for relief. Naturally those headaches caused by meningitis, brain tumors, etc., are not in this category, although there are some causative relations to the mechanism of the pain. Not many years ago chronic headaches in most of the patients were looked upon and treated as sinus headaches or other nasal conditions. A slightly thickened mucous membrane in the nose was considered a chronic hyperplastic sinusitis and the cause of recurring pain. A deviation of the nasal septum was the explanation for reflex pain in the head. Many extensive sinus operations were performed —septum resected, turbinates removed— and patients treated for months and years on the basis that sinusitis was the cause of their difficulty. To be sure, there still are cases of acute suppurative as well as chronic suppurative and allergic sinus membranes which could cause head pains, but the percentage is low. In recent years more logical and more demonstrable causes are presented. These are based on vascular behavior in the head. The work of Wolff [11] and others has demonstrated experimentally and clinically that the vessels in the cranium are responsible for most chronic headaches, and the pain of intracranial growths and acute inflammations is also a manifestation of the actions of these conditions on the cranial vessels. The pain impulses from the vessels are conveyed by the fifth nerve and perhaps the great superficial petrosal of the seventh nerve (Gardner, Stowell, and Dutlinger [12]) in cases of pain in the front part of the head; the glossopharyngeal, vagus, hypoglossal cranial nerves, and the upper three cervical posterior sensory roots in the posterior part of the head carrying these impulses from the vessels below the tentorium. The mechanisms are displacement of the large venous sinuses and traction on the veins entering into them; traction on the middle meningeal artery; torsion or displacement of the large arteries of the circle of Willis; distension and dilatation of intracranial arteries; inflammation in or about sensitive structures within the head, and direct pressure by tumors on cranial and cervical nerves conducting afferent fibers from the head to the brain. The same mechanism prevails when the extracranial vessels are involved. The vascular system being so deeply involved, it is easy to understand the influence of the autonomic system in this respect owing to its influence on vascular function.

The types of headache one encounters are tension headaches, migraine, allergic headaches, and various forms of atypical facial neuralgias. The tension headaches are usually found in persons with emotional problems, particularly such as affect their own personal comforts, dangers and problems. These headaches are usually associated with cervical and occipital muscle tension and tenderness. They may be located in any part of the head and face, are not lancinating, but the sensation is one of constant pressure and gnawing. A major factor in the mechanism is the vascular dilatation of the external cranial vessels. The headaches

usually disappear when the patient's problems disappear, or they may be relieved by vasoconstrictors like ergotamine with caffeine (Cafergot) or some of the variations of this preparation.

Migraine is a headache with similar mechanisms. There may first be a constriction of the extracranial or intracranial vessels followed by dilatation. It usually exists in persons with a constant drive to accomplish things, and a number of persons in their family are subject to these spells. Because of the onset with vasoconstriction, scintillating scotomata may be present at the onset. It is usually a hemicrania but may assume a sensation of general pressure in the head. It finally goes on to vascular dilatation and is self-limiting, often to recur in the near future when problems appear not to the patient's liking. This, again, is subject to relief by vasoconstrictors of the ergotamine tartrate group, further illustrating the vascular component.

Histamine cephalalgia is an allergic manifestation of the vasoconstrictor mechanism with venule dilatation and retrograde pressure of venous blood, vascular permeability, histamine effusion into the blood, and edema. This is a lower-half headache, lancinating, sudden in onset, radiating from the occiput to the face and neck, and is associated with lacrimation. It is similar to the pain previously described as sphenopalatine ganglion neuralgia. This headache seems to be amenable to histamine desensitization.

The neuralgias, including the sphenopalatine ganglion syndrome, are now looked upon as a manifestation of misbehavior of the extracranial vessels. The latter syndrome is believed to be due to a dilatation of the internal maxillary artery. The previous good results reported by sphenopalatine ganglion injection are explained on the basis that incidental sensory fibers which pass through the ganglion were the reason for relief. The ganglion itself is not considered to have any sensory component but is purely parasympathetic, controlling secretion and autonomic vascular motor function.

Conclusion

In conclusion, it may be said that when one reviews the number of conditions one meets in otolaryngology that are dependent on the vascular mechanism of the cranium, one is compelled to think in terms of these vessels as a group whose dysfunction produces symptoms in our field. In thinking along these lines, we are more likely not to overlook instances for which this is responsible and attribute the patient's illness to some other nonrelated condition.

REFERENCES

1. Murphy, J. P.: Cerebrovascular Disease, Chicago, The Year Book Publishers, Inc., 1954, p. 140.

2. Murphy, J. P.: Cerebrovascular Disease, Chicago, The Year Book Publishers, Inc., 1954, p. 131.

3. Hicks, S. P., and Warren, S.: Infarction of Brain Without Thrombosis: Analysis of 100 Cases with Autopsy, A. M. A. Arch. Path. 52:403-412, 1951.

4. Pratt-Thomas, H. R., and Berger, K. E.: Cerebellar and Spinal Injuries After Chiropractic Manipulation, J.A.M.A. 133:600-603, 1947.

5. Alvarez, W. C. The Neuroses, Philadelphia, W. B. Saunders Company, 1951.

6. Alvarez, W. C.: Cerebral Arteriosclerosis with Small, Commonly Unrecognized Apoplexies, Geriatrics 1:189-216, 1946.

7. Murphy, J. P.: Cerebrovascular Disease, Chicago, The Year Book Publishers, Inc., 1954, p. 207.

8. Dandy, W. E.: Intracranial Arterial Aneurysms, Ithaca, N. Y., Comstock Publishing Co., Inc., 1944.

9. Williams, H. L.: Ménière's Disease, Springfield, Ill., Charles C Thomas, Publisher, 1952.

10. Fowler, E. P., Jr.: Capillary Circulation with Changes in Sympathetic Activity: Blood Sludge from Sympathetic Stimulation, Proc. Soc. Exper. J. Biol. & Med. 72:592-594, 1949.

11. Wolff, H. G.: Headache and Other Pain, New York, Oxford University Press, 1948.

12. Gardner, W. J.; Stowell, A., and Dutlinger, R.: Resection of Greater Superficial Petrosal Nerve in Treatment of Unilateral Headache, J. Neurosurg. 4:105-114, 1947.

The Wire Fixation of Autogenous Bone Grafts in the Difficult Rhinoplasty

A Study in Osteogenesis

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Introduction

It is the purpose of this paper to show that the insertion and fixation of an autogenous bone graft is a valuable adjunct in the repair of certain nasal deformities. It is not intended to suggest such a procedure as a routine in all cases. Unfortunately, a surgeon often feels that his own "routine rhinoplasty" answers most, if not all, nasal plastic problems.

In a recent article Cottle[1] stated: "Technically, I suppose, the greatest single thing which the rhinologist has learned and taught in this work is that a rhinoplasty is not a hard and fast routine thing."

The repair of defects in bone, generally speaking, by the application of an autogenous bone graft is a gratifying procedure in most instances. The success of this operation depends to a great extent on the surgeon's knowledge of bone growth, coupled with his ingenuity, and the response of the individual patient's tissue to injury.

History

The recent exciting advances in grafting homogenous tissue are daily becoming history. The autogenous grafting of bone is already a definitely accepted procedure, yet Ollier[2] in 1867 was the first to publish his work on bone grafting. Macewen substantiated this work, and with his publication bone grafting became an established procedure. Sir Harold Gillies was one of the first to use bone in restoring nasal bridge contour. Then, in 1941, Mowlem[3] stated that about 25% of cartilage autografts became distorted. He used autogenous cancellous bone in preference to cartilage grafts and felt that survival depended on early permeation by tissue fluids. In discussing this question before the Congress on Wound Healing in Cambridge in 1943, Mowlem stated that all bone grafts could be divided into two groups: those which died and were replaced, and those which lived. Peer[7] has more recently stated that "an increasing number of investigators feel that the survival of autografts depends on the survival or death of their parenchymal cells."

Regardless of the fate of the cells and the method of survival, the autogenous bone graft is today an accepted procedure in nasal corrective surgery.

Experimental Work

"There is no such tissue in the body which is capable of so much overgrowth, or on the other hand, so much absorption as bone."[10]

For 200 years, as Ham[4] points out in an excellent review of the subject, there has been a difference of opinion as to whether new bone is formed by periosteum or by bone cells. He states rather conclusively from his experimental evi-

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dence that the source of new bone arises from the inner layer of the periosteum, the so-called osteogenic cell layer. This layer also lines the Haversian canal system. This theory states that osteogenic cells divide by mitosis into two layers, the inner layer becoming the bone cells, which, in turn, become surrounded by bone. Bone does not expand when once formed and can increase only by means of new bone deposited on one of its surfaces. Capillaries must accompany this bone growth.

Other investigators suggest that, as a result of metaplasia, new bone can form in a defect from ordinary connective tissue. Still others feel that the cells of bone or the osteocytes can form new bone.

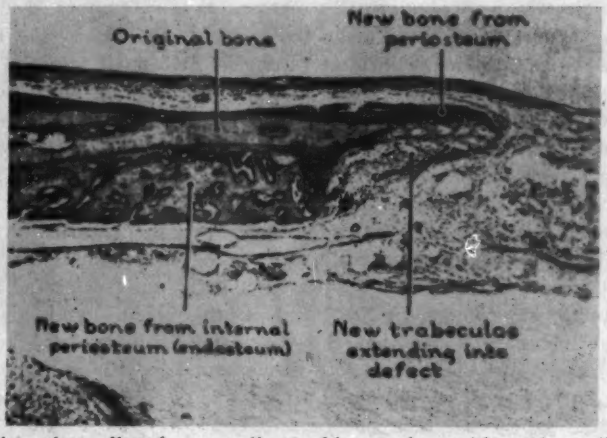
Original bone
New bone from periosteum
New bone from internal periosteum (endosteum)
New trabeculae extending into defect

Fig. 1.—A photomicrograph of the repair of a skull defect in a rat. Note the dead osteocytes, or bone cells, in the empty lacunae. Repair of the defect by osteogenic cell proliferation from three sources may be observed.

During a recent research project I made an effort to inhibit osteogenesis by creating defects in the cranial bones of rats. In these experiments evidence conclusively supported the first theory—i.e., the osteogenic cells of the periosteum, the endosteum and the diploic lining proliferated and gave rise to cancellous bone. The bone cells died in the bone approximate to the defect, as evidenced by the empty lacunae, but the calcified matrix remained to be slowly encircled by new healthy bone from the osteogenic cell layer. The matrix was gradually replaced by new bone (Fig. 1), and the word "gradually" is emphasized; this replacement is a slow process. In none of over 200 animals operated upon did metaplasia of fibrous connective tissue evidence itself and produce bone. Any free chips lying in soft tissue died and were absorbed by fibroblastic activity, similar to a foreign-body response. An occasional chip with osteoblasts on the surface survived if it approximated a blood supply. This fact was observed by Ham and Gordon[5] in dogs where cancellous iliac bone transplanted into muscle survived and new bone formed from the covering endosteal cells. This did not occur in thrice frozen and thawed cancellous chips and would tend to disprove the work of Levander[6] and others who have described the formation of new bone in soft tissues from connective tissue sources. There are claims[6] of producing bone by injecting into soft tissue cell-free extracts prepared from bone. It seems more practical at the present time, even if this is so, to use cells that produce bone, rather than to try to stimulate connective tissue cells that are disposed to form other types of tissue.

These animal experiments are suggestive evidence as to the fate of cancellous bone grafts and indicate the method whereby the graft survives or dies. By the application of these observations one

can more clearly visualize the histology of bone and apply these principles to the best of one's knowledge.

Choice of Material for Bone Grafts

Many leading surgeons agree that the availability and reliability of autogenous bone from the iliac crest surpasses all others in the repair of defects in the human nose. It is not the purpose of this paper to argue against the use of homogenous bone and foreign-body material, which everyone admits may have certain specific applications in a few situations. The intercellular substance of bone has a system of canaliculi through which tissue fluid normally permeates from the surface capillaries to nourish the osteocytes in the lacunae. Bone is a vascular tissue and therefore cannot survive if far removed from a capillary. Most bone cells are not more than 0.1 mm. away from a capillary. However, when compact bone is used as a graft there are few, if any, surface osteoblasts left for setting up new centers of ossification. Peer [7] has pointed out after many human experiments with nasal septal and turbinate bones that they survived autotransplantation into soft tissue and that the osteocytes remained alive. This would suggest that, in this type of bone, tissue fluid nourishment alone sufficed to preserve the graft. In this connection drying-out of the graft certainly does not assist living tissue to survive; immediate transplantation to the recipient site is essential.

Compact bone, generally speaking, adds strength to a graft. It presents a smooth, even surface suitable to the profile contour of the nose. Although, sooner or later, compact bone is eventually replaced by new bone, it serves as a stimulant and conductor of osteogenesis, meanwhile carrying out the function for which is was intended.

Many have found that removal of periosteum does not influence the graft. It might be pointed out that after simple elevation of periosteum by stripping it from the iliac crest the covering osteogenic cells are often left behind on the cortical bone and can be transplanted with the graft. The osteoperiosteal implant protects the graft from rapid revascularization and so slows absorption, and if converted to scar tissue it can warp the underlying graft, producing distortion of the nose.

Cancellous trabeculae are covered with endosteum which, as stated previously, has osteogenic qualities. A point to remember is that these cells, if they live, protect the bone from resorption. Epithelium in contact with bone will slowly dissolve the adjacent bone. This is seen in tumors and in cholesteatoma formation in the temporal bone; therefore almost any cell is potentially capable of etching and resorbing unprotected bone.[8] When this type of bone graft is placed in the nose, against the nasal bones and frontal bone, a blood supply is presented to maintain the osteogenic cells covering the exposed graft surface. This allows osteoblastic activity to proceed, and union with the host is possible. Recently Kiehn and his associates [9] found that radioactive isotopes, injected systemically, were present in an autograft 24 hours after operation. They felt that one could consider such a graft a firmly established transplant after 15 days. This suggests free interchange of tissue fluid in a freshly transplanted autograft. In this experiment no isotope was found in homogenous transplants. Thus a cancellous graft becomes more rapidly vascularized than a compact bone graft, a fact that is readily understood when the relative difference in blood supply is considered.

The question of fibrous union versus osseous union of the graft with the host is difficult to answer authoritatively, as observers differ. In Figure 3 it would appear that fibrous union is present, but close examination shows bony union, and clinical manipulation substantiates this observation.

Type of Cases Suitable for Grafting

When the dorsal line is depressed by a congenital deformity or disease, or deflected and depressed by trauma, the nasal bones, the cartilaginous vault, and the septum may all be involved. Following trauma it is not too difficult to restore these elements to the midline after the septal deformity has been removed, but to maintain them in this position and provide a dorsal contour line is a problem. The insertion of a bone graft tends to anchor these structures in a desired position. It is admitted that no procedure gives consistently good results, but the method to be described is a step in that direction. When cartilage is involved, the support of the tip is decreased and the common lip-columella angle deformity occurs, with scarring producing a depressed nasal tip. As stated previously, the septum must first be repaired by a submucous resection to obtain an adequate airway. To quote Hilger,[11] "The problem of architectural nasal obstruction has largely resolved itself into the proper surgical correction of the anterior third of the nose and septum. Rhinoplastic surgery has a physiological as well as a cosmetic responsibility." As far as I know, Cottle concurs in this opinion. From the point of view of appearance in these cases, a long graft is preferable to a short one which cannot be controlled in position, nor may a final result be predicted. For example, if a lateral osteotomy does not give sufficient projection after refracture, a wide nose with a broad pyramid can be narrowed by the use of an onlay iliac graft. Many surgeons are able to carry out a transurethral prostatectomy as well as, or better than, others performing a suprapubic procedure. A similar situation exists in the method of approach to the correction of the broken nose.

Method and Discussion

The routine of taking a bone graft from the iliac crest and the method of insertion in the nose are clearly described by Foman.[12] I remove the iliac graft through an elliptical incision 1 in. below the crest to afford postoperative drainage. The scar is thus below the pressure point and will be less painful. The periosteum is lifted from the crest with a mastoid periosteal elevator. Sterile wooden applicators are placed in the nose in the exact position of the grafts and carefully cut to the desired lengths; these can then be used over the crest to measure the amount of exposure necessary. The grafts are cut in situ, reducing the amount of modelling necessary before insertion. Cortical bone is included in the graft for stability, and the cancellous bone can be shaped by bone-cutting forceps and a rasp. Dental burrs are not used. Converse[14] shapes the dorsal graft so that it is broader over the nasal bones, thus resembling the normal nose from a front, full-face view; the normal curvature of the iliac crest gives the postoperative profile a pleasing effect. The columella graft of course must be narrower and is also measured and notched to receive the dorsal graft, thus avoiding a beaked appearance which may occur from an overriding dorsal graft (Fig. 2).

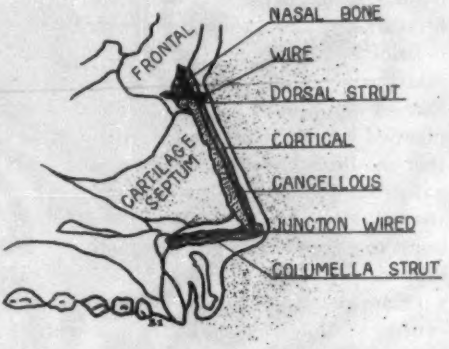

Fig. 2.—A diagram to illustrate the position and method of fixation by use of two autogenous iliac crest grafts.

The incisions are made either in the columella, as described by Brown and McDowell,[13] a method which I advocate,

or through previously made intercartilaginous incisions extending down the border of the medial crus. If one contemplates suturing of the two grafts, the procedure is made easier by the former approach. The closure of the midcolumella incision must be carried out as a plastic effort, with careful approximation of skin edges, otherwise pits and scarring occur. This latter situation may be avoided by leaving sufficient subcutaneous tissue to allow accurate approximation. The nylon (Dermalon) skin sutures are removed on the fourth postoperative day. A transverse incision is difficult to revise at a later date, owing to scarring, and should be avoided.

Prior to insertion of a graft, a rhinoplasty and submucous resection may be carried out. These procedures are planned so that the graft will not be just a thick overlay, obliterating the nasofrontal angle. A groove or a cube-shaped notch must be made in the frontal bone to receive the graft, which can be tapped into this fixation point. Again, if a rhinoplasty has not been performed a central section of nasal bones and septum is removed with a chisel or saw to form a bed for the graft. A small hole is drilled in the dorsal and columella grafts so they can be united and fixed under the lobule with fine steel wire. Some surgeons use absorbable surgical (gut) sutures but the tensile strength of this material disappears with absorption, and a slip at this point can cause a lateral deviation of the columella and nasal tip.

If excess tension of the skin over the graft is present it is necessary either to undermine further or to thin the graft, with reduction in the dorsal projection. Any irregularities of the graft surface are smoothed or removed to facilitate easy insertion, particular attention being paid to the columella strut. A small notch is made in the maxilla at the nasal spine to receive the columella graft and prevent slipping. The period of time from removal of the graft from the crest to insertion should be as short as possible to avoid drying of the cells; perhaps the modelling should be carried out in Ringer's solution. When the grafts are in position and wired together, a 0.035 in. Kirschner wire is introduced after the method of Farmer* through the skin and the dorsal graft, and is driven into the nasal process of the frontal bone for ¼ in. The wire is then nipped off at the skin surface and can be buried by manipulation of the skin. This wire is removed at three weeks, or sooner if infection at the point of entry is noted. In a few cases it has been left in permanently (Fig. 4). Primarily, the aim of this fixation wire is to maintain contact of the graft with the host, and, secondly, to prevent downward slipping with the production of a beak tip and to prevent scar tissue from depressing the nasal tip in the healing stage. On palpation the graft feels firm, and the surgeon has confidence that if slight trauma occurs, for example on recovery from general anesthesia, displacement is less likely to occur. This pin is checked for position, as is the graft, by radiography studies on the fifth day postoperatively. The x-ray in Figure 3 was taken six weeks after operation and shows the graft to be more radiolucent than the surrounding bone, suggesting a change in vascularity. Small bone grafts are easily displaced, and without stress being present they tend to become absorbed. Foman [15] feels that two large grafts are preferable to single small grafts and that L-shaped grafts are undesirable. An effort is made to obtain as close a contact with the host as possible, although this may be difficult when scar tissue is present in old fracture cases (any loose bone or scar can be removed under direct vision). Bleeding is controlled prior to insertion of the graft either by pressure from without or by using warm saline or epinephrine solu-

* Farmer, A. W.: Personal communication to the author.

Fig. 3.—From left to right: Passport photograph before injury. Profile before graft operation. Radiographic study of the fate of the graft three months after operation. Profile postoperatively.

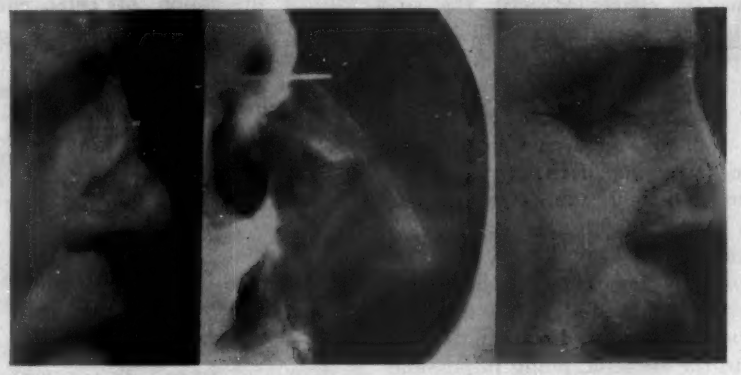

Fig. 4.—Repair of scar contraction from atrophic intranasal changes. The insertion of the graft was associated with repair of the septal defect with use of bank cartilage and a cancellous plate.

tion. The formation of hematoma is an undersirable complication, since it predisposes to scarring and deformity of the graft with subsequent absorption. If the graft is displaced or unsatisfactory when the first dressing is carried out, the patient is taken to the operating room, where the clots are removed and the graft again immobilized and a firm pressure dressing applied. For this reason, the first complete dressing is carried out on the fifth postoperative day, before the patient becomes eager to go home. Unnecessary haste in removing the petrolatum packing, if a rhinoplasty has been performed, will pull down the upper lateral cartilages, produce bleeding, and necessitate repacking. If an overlay graft without rhinoplasty has been performed, no internal dressing is required. The technique of Straith [10] has been found useful in maintaining the septum in the midline where dorsal support is necessary from within; here a pin is inserted beneath the perichondrium to the frontal bone and anchored to a columella pin. However, combined with a bone graft, this makes the operation rather tedious. Usually internal packing will suffice if the nose has been broken down into its components and then remodelled in position.

External fixation should securely anchor the splint to protect the grafts from injury. The splint must extend down to and over the nasofacial groove to pre-

vent, if possible, hematoma formation in this region. In some patients this splint may be removed on the fifth day, being replaced for sleeping only.

General Comment

A method is described for repairing a depressed and crooked type of nasal deformity. Once the cartilaginous support is removed, the surgeon is forced to build up the dorsal line both for function and appearance. A one-stage procedure may have a drawback in the consideration of functional and cosmetic results obtaining equal results. However, this paper is concerned with the situation wherein breathing cannot be improved properly without a repair of the external deformity.

It has been emphasized that one cannot fill in a small depression alone, but, with the narrowing of the pyramid and reduction in height, the defect can be filled in with an adequate graft. This graft must be fixed in the midline so that close approximation of host and graft is possible, and it is felt that pin fixation is a step in this direction. Nature is kind to the surgeon, filling in possible gaps between graft and host either with new bone or by fibroblastic activity with accompanying blood vessels. A bone graft with its slight irregularities tends to add character to the profile!

The midcolumella incision has fallen into disfavor; however, it is an easier approach, and closure without visible scar formation is possible in most cases.

In children iliac grafts are not suitable material, since they are cartilaginous and their removal may interfere with proper development of the crest. Here, then, one must use cartilage grafts, with a less perfect result to be expected.

It is always sound practice to take preoperative photographs and to observe a case at length before beginning this type of repair. Occasionally, with a marked deviation of the septum, one must proceed in two stages. During the first stage scars may be removed from the nose, and perhaps narrowing of the pyramid may accompany the submucous resection. In this way a more perfect result may be expected after insertion of the bone graft. Most noses requiring this type of rhinoplasty also require a submucous resection, and the results enhance one another.

One cannot lay down generalizations for all cases; each case must be considered separately.

Summary

A method of fixation of an autogenous iliac bone graft is described, together with a discussion of osteogenesis as applied to the subsequent fate of such bone grafts.

Dr. A. W. Farmer conceived the idea of pin fixation and demonstrated the usefulness of this procedure; Mr. Arthur Smialowski, the photographer, Photographic Department, St. Michael's Hospital, Toronto, made the photographs, and Mrs. Florence Smith performed the secretarial work.

REFERENCES

1. Cottle, M. H.: The Role of the Rhinologist in Rhinoplasty, Laryngoscope 63:608-618 (July) 1953.

2. Ollier, L.: Traité expérimental et clinique de la régénération des os et de la production artificielle du tissue osseux, Paris, V. Masson & fils, 1867.

3. Mowlem, R.: Bone and Cartilage Transplants, Brit. J. Surg. 29:182-193 (Oct.) 1941.

4. Ham, A. W.: Cartilage and Bone, in Special Cytology, by E. V. Cowdry, Ed. 2, New York, Paul B. Hoeber, Inc., 1932.

5. Ham, A., and Gordon, S.: The Origin of Bone That Forms in Association with Cancellous Chips Transplanted into Muscle, Brit. J. Plast. Surg. 5:154, 1952.

6. Levander, G.: A Study of Bone Regeneration, Surg. Gynec. & Obst. 67:705, 1938.

7. Peer, L. A.: Autogenous Bone Transplants in Humans, Plast. & Reconstruct. Surg. 13:56, 1954.

8. Ham, A. W.: Histology, Philadelphia, J. B. Lippincott Company, 1950.

9. Kiehn, C. L.; Gukentag, J., and Glover, D. M.: Localization of Isotopes in Bone Grafts by Autoradiography, Plast. & Reconstruct. Surg. 14: 425, 1954.

10. Boyd, W.: A Textbook of Pathology: An Introduction to Medicine, Philadelphia, Lea & Febiger, 1953.

11. Hilger, J. A., in Teacher's Section: Otolaryngology, Tr. Am. Acad. Ophth. 56:500, 1952.

12. Foman, S.: Surgery of Injury and Plastic Repair, Baltimore, Williams & Wilkins Company, 1939.

13. Brown, J. B., and McDowell, F.: Plastic Surgery of the Nose, St. Louis, The C. V. Mosby Company, 1951.

14. Converse, J. M.: Technique of Bone Grafting in Contour Restoration of the Face, Plast. & Reconstruct. Surg. 14:332, 1954.

15. Foman, S.; Goldman, I. B.; Neivert, H., and Shattner, A.: Rhinoplastic Aphorisms, Reflections and Paradoxes, Eye, Ear, Nose & Throat Month. 31:537, 1952.

16. Straith, R. E.; Teasley, J. L.; von Linde, M. G., and Moore, L. T.: The Treatment of Lateral Deviations of the Nose by Pin Fixation, Plast. & Reconstruct. Surg. 15:346, 1955.

Osteoma of the Tongue

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True osteomata of the tongue, comprised solely of dense bone, are very rare lesions. In fact, tumors of the tongue containing bone are almost as unusual as osteomata. When one considers that the tongue is an active muscular organ subject to trauma, especially at its tip and margins, the relative lack of calcified tumors is surprising.

A review of the world's literature reveals a total of eight cases since Monsarrat[1] published the first case report, in 1913. An asymptomatic lesion the size of a small cherry was accidentally discovered in a 32-year-old woman. The tumor was pedunculated, freely mobile, and located 2 to 3 mm. lateral to the "sulcus terminalis." The mass was easily removed. Microscopic sections revelaed a regular, dense, bony tissue with a few narrow Haversian canals. The bony lamellae formed a circular or oval pattern with only a few osteocytes.

Hirsch[2] reported the second case, in 1925. This osteoma was found on the dorsum of the tongue in a 26-year-old woman who complained of some difficulty in swallowing. A wire snare was used for the extirpation of the mass. From a histologic standpoint, it resembled the first case in that it was comprised of dense bone.

Zuckerman[3] recorded the third osteoma of the tongue, in 1929. It was found coincidentally in a 30-year-old woman who had a right maxillary sinusitis and a polyp of the right middle meatus. She complained of a foreign-body sensation at the base of the tongue after nasal surgery had been accomplished about a month prior to the complaint. A pea-sized, pedunculated mass was found, arising to the left of the midline in the area of the circumvallate papilla. This mass was excised, and the base was cauterized. Histologically, the lesion was composed of bone with Haversian canals and marrow spaces.

Jung[4] reported a case study of a 22-year-old woman who complained of throat irritation. Small, firm, elevated nodules were found to the right and left of the foramen caecum. The nodules were excised under local anesthesia with little bleeding. They were smooth and bony hard and were found to contain bony tissue, Haversian canals, and marrow spaces. A fibrous capsule was noted about each lesion.

The fifth case of osteoma of the tongue was published by Magnien and Perrot.[5] A 37-year-old woman complained of some disturbance in swallowing. Examination revealed a painless mass of the size of a large pea located at the juncture of the posterior and middle third of the tongue. It was sessile and pale pink in color. Histologic study was again similar in revealing a picture of adult compact bone with medullary spaces and Haversian canals.

Muta and Ogata[6] described a sixth osteoma of the tongue, in a 23-year-old man who had had four months of some dysphagia and a previously noted tumor of the tongue. The lesion was found just to the right of the framen caecum and was pedunculated, measuring 0.6×0.5×

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0.4 cm. It was bony hard on palpation. The mass was found to contain cancellous bone.

The seventh osteoma was reported by Witchell [7] in 1949. A 30-year-old woman patient was seen because of headache, postnasal discharge, and a tendency toward gagging. Six days prior to examination, she had palpated a mass in the back of her throat. Examination was negative save for a rounded mass about 1 cm. in diameter attached to the middle of the right half of the circumvallate papilla by a short, broad pedicle. Excision was easily performed under local anesthesia with little bleeding. The pathologist reported that the mass was composed of compact bone of normal architecture enclosing a few marrow spaces.

The most recent case was described by Breckenridge and Lukens.[8] This was a 23-year-old woman who had been aware of a growth at the base of her tongue since infancy. The mass was attached just to the right of the midline in the area of the circumvallate papilla. It was about 1 cm. in diameter and pedunculated in type. Removal was easily accomplished with scissors. Histologically, the tumor was composed of mature cancellous bone and small Haversian canals.

Etiology

With only a few cases to consider, it is not surprising that pathogenesis of osteoma of the tongue is somewhat obscure. In fact, some of the authors have not even attempted to discuss etiologic factors. Other authors have variously ascribed these osteomata to a malformation formed by ossification of branchial arch remnant,[1] epignathus of an atypical site or ossified accessory struma,[2] congenital heterotopic tumor,[3] ossified heterotopic germ of branchial or mandibular arch,[4] and ossification of the lymphatic tissue of the blind canal of the tongue.[6] Greig [9] theorized that these osteomata arose through a complex degeneration of a fibroma of the posterior third of the tongue.

Report of Case

A 27-year-old Negro woman was seen on May 19, 1955, with the complaint of being "somewhat choky and nauseous." A mass on the tongue was

Fig. 1.—Histologic examination revealed a central area of cancellous bone with small Haversian canals and a few marrow spaces. This is encapsulated by a zone of fibrous tissue. Peripheral to this is a layer of stratified squamous epithelium. Reduced about 1/5 from mag. × 15.

Fig. 2.—High-power view of osteoma showing Haversian canals. Reduced about ½ from mag. × 65.

first noted about five months previously. Examination was negative save for the tongue findings. A yellowish to grayish, firm, elevated mass about the size of a green pea was noted just to the left of the median line at the level of the circumvallate papilla. The mass was easily removed with a cold snare. The slight bleeding was controlled with a silver nitrate stick. The operative diagnosis was that of fibroma. The pathologist reported a mass measuring $0.7 \times 0.6 \times 0.3$ cm., glistening gray in color and of a stony hard consistency. Histologic examination revealed a central area of cancellous bone with small Haversian canals and a few marrow spaces. About this is a zone of fibrous tissue. Peripheral to this is a layer of stratified squamous epithelium (Fig. 1). The pathologic diagnosis was osteoma.

It is of interest that the patient has become asymptomatic since the surgery. Routine urine and blood studies, blood calcium and phosphorous, and x-rays of the skull and long bones have all been entirely negative for pathologic variations.

Comment

A total of nine cases of osteoma of the tongue, including the present report, are now to be found in the literature. All save one were in women. Only once were two lesions noted in the same patient, and that was found at the initial examination. All lesions were seen in persons of 20 to 40 years of age, and all were found in the posterior third of the tongue, usually in fairly close relationship to the foramen caecum. It is noteworthy that one patient gave a history of a growth being noted at the back of the tongue during infancy.

We are therefore of the opinion that these osteomata of the tongue undoubtedly have a congenital basis for their origin, since all have been found near the foramen caecum. They may be a congenital remnant of a branchial arch with ossification to form cancellous bone.

The tumors are bloodless, small, benign, and painless, and usually asymptomatic. They are easily removed by scissors or snares.

Summary

The ninth case of osteoma of the tongue is reported. We believe that its presence is best explained on some congenital basis, with or without later ossification.

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REFERENCES

1. Monsarrat, M.: Ostéome de la langue, Bull. et mém. Soc. anat. Paris, 88:282-283, 1913.

2. Hirsch, O.: Knochentumor am Zungengrund, Monatsschr. Ohrenh. 59:496, 1925.

3. Zuckerman, M.: Ein Fall von Osteom der Zunge, Ztschr. Hals-Nasen-u. Ohrenh. 25:118-120, 1929.

4. Jung, G.: Über ein Osteom der Zunge, Beitr. klin. Chir. 154:167-168, 1931.

5. Magnien and Perrot, M.: Un cas d'ostéome de la langue, Ann. d'anat. path. 10:331-334 (March) 1933.

6. Muta, L., and Ogata, K.: Ein Fall von Osteom am Zungengrund, Oto-rhino-laryng. (Japanese) 11:1016-1017 (Nov.) 1938.

7. Witchell, I. S.: Osteoma of the Tongue, Arch. Otolaryng. 50:453-456 (Oct.) 1949.

8. Breckenridge, R. L., and Lukens, R. M.: Osteoma of the Tongue, Arch. Otolaryng. 52:19-24 (July) 1950.

9. Greig, D. M.: Osteoma of the Tongue: Critical Review, Edinburgh M. J. 39:93-104 (Feb.) 1932.

Audiology Section

JOSEPH SATALOFF, ASSOCIATE EDITOR

The Use of Hearing Aids for Severely Deaf Children

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In this discussion of the problem of the use of hearing aids it should be understood that the children referred to here are severely and profoundly deaf. That is to say, they are children who, as a result of the onset of deafness before or immediately after birth, or during early infancy, are unable to acquire speech and language naturally unless special help is given. Obviously children whose handicap is less severe will benefit very much from the use of aids, but in their case the therapy is more obvious and the results well established.

Preschool Children

Until seven or eight years ago the use of hearing aids by small children was not thought desirable. In fact, until the development of more compact and lighter aids it was not practicable, since no infant would find pleasure in being encumbered by bulky and heavy apparatus. This point is an important one, since the aid needs to become, almost from the beginning, an integral part of the child's clothing and equipment. He needs to learn to grow up with it, taking for granted the benefits derived and accepting the minimal way in which it makes him different from other children because the advantages of using it outweigh the disadvantages.

In the clinic attached to the Department of Education of the Deaf at Manchester University where the hearing of preschool children is tested and where their parents are given guidance about home training, increasing use has been made over the past seven years of hearing aids for very young severely deaf children. Recent work by Miss A. M. Pickles (Alexander Pigott Wernher Research Fellow) in this clinic has shown the value of this approach in the mental and social development of the children.* The youngest children in whom the aids have been used were aged 11 months, and all of them under 3 years. It will be evident that if use is to be made of residual hearing at such an early age there must be adequate provision for diagnosis. Tests for ascertaining deafness in infancy have been available for a considerable number of years,[2] but the problem is rather one of ensuring that children are brought for testing at a sufficiently early age. Dependance for this has generally been placed on the parents' suspicion of deafness in cases where there are no obvious indications, such as congenital abnormality or illness. It is not often that the parent or general practitioner suspects deafness until after the normal age of talking, so that it is much less frequent than is desirable that referrals for clinical examination are made before the age of 2 years, although most pupils admitted to schools for the deaf have become deaf before the age of 2 years. In order to meet this problem and provide a means of screening out infants with defective hear-

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*An account of this work is to appear in Ewing.[1]

ing, a scheme has been devised whereby selected health visitors belonging to the Maternity and Child Welfare Service can make screening tests of hearing of young children either in their own homes or at child welfare clinics. The inspiration behind this scheme has been Dr. I. R. Ewing, who has herself trained a very large number of these health visitors to make the tests and has helped in the establishment of diagnostic clinics under a number of local authorities. Children who fail in these screening tests are referred to the diagnostic clinics, where a more detailed program of testing is carried out under the direction of a member of the staff of the medical officer of health (with guidance in the early stages by Dr. Ewing) and where parents are given guidance about the appropriate home training that should be given to their child. The work of the first of these clinics has been described by Humphreys.[3]

When it has been ascertained by diagnostic testing that there is some useful residual hearing, even though this may not amount to a great deal, a hearing aid is recommended. At first the child is introduced to the aid by the clinical worker in the presence of the parents, and the latter are shown how the child can be helped to use it most effectively. Not until the parents show that they can make satisfactory use of the aid are they encouraged to continue with the work at home. Only short periods, not exceeding five minutes in duration, are advised at first, but the frequency of such periods during the day should be as often as the parents can arrange. Progressively the periods are lengthened until the child is willing to wear the aid continuously (in some children often from the age of 18 months).

This work, and experiments conducted by Prof. G. W. Hardy at The Johns Hopkins University, raises new educational problems. Some of these severely deaf children are already beginning to learn speech and language through this combination of aided hearing and watching before the age when they can be admitted to a school for deaf children. Will, in fact, this mean that they do not need to go to special schools for deaf children? Our evidence is that most of these pupils will still need full-time special help from teachers qualified to teach deaf children. In some cases no decision can be made until the child has had a prolonged period of this training at home and until he is almost 5 years of age. In a limited number of cases children with losses over 80 db. after a number of years of good training under expert guidance can talk and comprehend speech sufficiently well by the age of 5 years to enable them to attend ordinary schools and hold the position there that their age and ability warrants. It is too early yet to be able to make a prognosis as to which children are likely to be able to follow a normal school curriculum. Innate ability, etiology, and the kind of guidance given are among the factors that need to be studied in this connection, but further experiments are required before useful conclusions can be drawn. Any children who are admitted to ordinary schools must be very carefully watched to ensure that the decision was a right one, and transfer to a special school should be made if the pupil does not continue to make the anticipated progress. For the majority of the children who need to attend schools for the deaf their attainments on entering school after having received this guidance provide a marked contrast to the wordlessness of the untrained child, while their attitude and social behavior displays a higher level of maturation.

Severely Deaf Pupils in School

Experiments in the Department of Education of the Deaf, Manchester University, over the last 20 years, described by Ewing, Ewing, and Littler,[4] Ewing and Ewing,[5] and by Clarke,† as well as more recent experiments at the Clarke School, in Massachusetts, described by Hudgins,[7] have shown

† Clarke, B. R.: An Experiment in Auditory Training with Profoundly Deaf Children, unpublished Ph. D. Thesis, in University of Manchester library. Also summarized in reference 6.

that severely and profoundly deaf pupils can gain very great benefit from the consistent and systematic use of hearing aids in schools for the deaf. These benefits include greater facility in comprehension, greater skill in the use of language, and more intelligible speech. Results are not obtained dramatically, as a rule, and are only achieved after long and arduous work on the part of both teachers and pupils.

The best arrangements seem to be that for pupils of 7 years of age and upward a group hearing aid should be provided for each classroom, so that the pupils can have continuous practice during all the lessons when watching and listening are appropriate. For pupils under the age of 7 this arrangement is somewhat too rigid and leads to a formality that is undesirable at this stage. These children would be better equipped with individual hearing aids, but in addition there should be provided for each class a mains-operated individual hearing aid (variously described as an auditory training unit, a speech-training hearing aid) so that the children have the opportunity of hearing speech reproduced with high fidelity at high intensity levels during periods of individual speech improvement. Pupils over the age of 7 would continue to use their individual hearing aids at appropriate times out of school, and further systematic training in their use would be given to the senior pupils, who would be dependent on individual aids as the only available reproducers of amplified speech when they left school.

The use of the above-named equipment in the classroom has, as an essential corollary, the provision of the best possible acoustic conditions. This does not mean that anechoic, completely sound-proof rooms are needed, but that sufficient treatment should be given to ensure that the level of external noise entering the room is low, that ambient noise generated in the room is also as low as possible, and that the treatment of the wall and ceiling surfaces reduces the reverberation time to a level at which maximum intelligibility of speech through the aid can be attained. Reduction of external noise can be achieved by the setting of the school block away from noisy thoroughfares, by ensuring that doors and windows are close-fitting, and by providing sound-insulation between rooms. Noise generated in the room can be reduced by the use of an appropriate floor covering as well as by padding or silencing the bottom of tables, chairs, desks, and other movable furniture. In regard to the reduction of reverberation, this is best done by treating a portion of the ceiling and walls with a sound-absorbent material, such as acoustic tiling. The amount of treatment depends to a large extent on the size and shape of the room, and to lay down the reverberation time that has to be achieved (say 0.5 seconds) is not helpful, since different effects can result from such a time in differently shaped rooms. It is usually recommended that about one-third of the wall and ceiling surfaces should be so treated, but the placement of the material is best done experimentally. At Manchester University we have been fortunate in being able to carry out experiments in acoustic treatment by the provision, through the generosity of the Council of the University, of a laboratory classroom. In originating the idea, Professor Ewing had in mind the value of being able to vary acoustic conditions experimentally for actual teaching purposes. Deaf pupils, therefore, taught in classroom conditions, as well as deafened and normal hearing adults, have been used as subjects in the experiments which have been carried out in this room by Mr. J. E. J. John, lecturer in audiology.

Besides the acoustic conditions in which it is to be used, the performance of the equipment is of considerable importance. Maximum intelligibility of speech is dependent on the intensity at which it is presented and the frequency response of the hearing aid. For pupils of the category with which we are here concerned it is necessary that the aid should be capable of presenting undistorted speech at the ear at

not less than 120 db. above normal threshold of detectability. When it is remembered that for persons with normal hearing speech needs to be at about 25 db. above their threshold of detectability before the threshold of intelligibility is reached,[8] then it will be appreciated that pupils who have losses over 90 db. (which can alternatively be described as threshold of detectability for speech at over 90 db.) require to listen to speech at upward of 115 db. above normal threshold if there is to be any likelihood of some of the speech being intelligible to them. The frequency response of the aid is usually governed by the response of the microphone and telephones. Although it has been widely suggested that equipment which shows a rapid fall-off in response after 3000-4000 cps is perfectly acceptable, recent work which has been done at Manchester University seems to indicate that better patterns of speech are given back by severely deaf children when the response of the aid is flat (or rising) up to at least 6000 and, if possible, 8000 cps. Microphones generally have a good response up to this figure, so the main limitations are usually in the receivers. Permaflux receivers do give a good response within this frequency range, but, so far, none of the miniature receivers which are available can do this. This seems to indicate that, at least in the meantime, Permaflux disc receivers are more suited for work with severely deaf pupils using group aids or speech-training hearing aids than miniature receivers with exact individual molds.

Two points arise in the use of aids which need very careful attention if their best results are to be achieved. The first of these is that incomparably better results are obtained when speech is uttered close to the microphone, (i.e., at a distance of about 9 in.). Since the input level should not exceed 70-75 db., only a quiet conversational voice is needed at this distance. The quality of the output is much better under these conditions than when speech is uttered further away, the effects of reverberation are considerably lessened, and the maximum output of the equipment can usually be achieved if it is required, so that the severely deaf pupils get the highest level which they can tolerate. This point is linked closely to the other, which is that these pupils need to be enabled to hear the sound of their own voices as well as those of other pupils and the teacher. The value of a hearing aid for speech improvement is diminished enormously if the pupil cannot hear the sound of his own voice. It is in the matching of his own patterns to those of the teacher that the greatest progress can be made in speech improvement. This necessitates his speaking close to the microphone, with the consequent provision of sufficient microphones in the group hearing-aid arrangement to enable this to be done. It is generally found that one microphone between two pupils is a satisfactory arrangement for this. Pupils have to learn to use the microphones effectively so that all members of a class can hear what is being said by the pupils as well as by the teacher. It will be evident that this can only be done satisfactorily when a group hearing aid is used, since, although pupils are likely to hear their own voices when wearing individual aids, they will have considerable difficulty in hearing the teacher or the other pupils at anything like a satisfactory loudness level unless the speaker can get very close to the microphone.

The need for long and arduous work on the part of pupils and teachers was stressed above. This, however, does not mean that time should be spent on periods of intensive listening practice. The concept of auditory training, in so far as it equates with specific exercises for the purpose of sense training, is not a particularly fruitful one. Twenty or thirty years ago pupils in schools for the deaf in this country spent a short time each morning in what were known as "breathing exercises." This was supposed to help them to develop good speech. While it is not denied that correct breathing is essential to good speech production, it has subsequently been shown that general bodily exercise, running, and games are just as effective in

obtaining the desired results. So it is with residual capacity to hear. Exposure to audible sounds, especially the sound of speech, for as long periods as possible is just as effective in enabling pupils to develop their capacity to hear as is a formal series of exercises designed for that purpose. Indiscriminate use of aids is not, of course, suggested, and the training must be planned to suit the age and stage of the pupil. Such planning must include listening in the best possible acoustic conditions, before listening in poor ones; it must include listening to one speaker only, before trying to concentrate on one voice which can be heard in a medley of other sounds and voices, and it must take into account the interests and capabilities of the pupils concerned.

With preschool children formal training is undesirable. The aid should be used for the purpose of listening to speech while watching the speaker. No advantage is gained, except for testing purposes, by any attempt to separate the two sensory pathways to the brain for the purpose of giving intensive training to one in isolation. The aim should be for the child to learn to comprehend speech in a way as nearly approaching that in which children with normal hearing learn to understand, that can be devised. This means repetition of the same phrases and sentences in appropriate contexts and repetition of the same words in different phrases and sentences so that, as with the hearing child, comprehension slowly emerges. Just as comprehension precedes expression with the hearing child, so we must expect deaf children to begin to understand before they begin to use sounds purposively themselves. Being able to hear the sound of their own voices through a hearing aid is of inestimable advantage in developing the capacity to talk by adding another dimension to the visual pattern which is seen on the face and the kinesthetic pattern which is felt when the speech organs are in movement.

This listening to and watching for speech will take place during all the play and the social and domestic situations of their daily lives. These are the natural situations in which language is developed, and it is through them that these severely deaf children will come to appreciate the social purpose of speech and language. Hearing, through their aids, other sounds of daily life will, of course, enrich their growing understanding both of language and of their environment.

When children enter school a similar pattern needs to be evolved. At the nursery and infant stages (that is, between the ages of 3 and 7) hearing aids should be worn for most of the normal group and class activities, being supplemented for individual speech work by the speech-training hearing aid. Hearing, however imperfect, is thus a normal accompaniment of the learning process and is not restricted to special periods for which the aid is donned. At the junior stage, when the children are normally more frequently engaged in class lessons, the group aid provides better listening conditions which can successfully be exploited. Once again, the aid should be thought of as providing the natural medium for contact between teacher and pupil and pupil and pupil. It is true that for such children lipreading will always be a major medium of communication, but the attitude of watching and listening should always be present in the minds of the children, developed by the skilled help and encouragement of the teacher. Senior pupils need to become clearly aware not only of the help an aid can give but of the specific limitations of that help in their own individual cases. They need to learn how to keep an individual aid in good working order and be able to get the maximum benefit from it under varying conditions. This will be an important part of the program in the senior department, so that when the pupils leave school they will want to continue to use the aid in adult life and not discard it as having greater disadvantages than advantages.

It should be evident from most of what has already been said that teachers of severely deaf children need adequate training

to make such a program effective. Although technical knowledge is not required, there must be a deep understanding of what the equipment is capable of and how it can be used to maximum advantage. It is not only that a new skill is required, but behind the skill there must be a new attitude, a new philosophy, which recognizes the potentialities of the pupils for this newer approach and has faith in the method as one which will lead the pupils to the maximum achievements of which they are capable.

The University of Manchester.

REFERENCES

1. Ewing, A. W. G., Editor: Educational Guidance for Children Handicapped by Deafness, Manchester, Manchester University Press, to be published.

2. Ewing, I. R., and Ewing, A. W. G.: The Ascertainment of Deafness in Infancy and Early Childhood, J. Laryng. & Otol. 59:309, 1944.

3. Humphreys, E. B.: Deafness in the Young Child, Med. Officer 91:39, 1954.

4. Ewing, A. W. G.; Ewing, I. R., and Littler, T. S.: The Use of Hearing Aids, Report 4, Medical Research Council, His Majesty's Stationery Office, 1936.

5. Ewing, I. R., and Ewing, A. W. G.: The Handicap of Deafness, New York, Longmans, Green & Co., Inc., 1938.

6. Clarke, B. R.: An Experiment in Auditory Training with Profoundly Deaf Children, Teacher of Deaf 52:11, 1953.

7. Hudgins, C. V.: The Response of Profoundly Deaf Children to Auditory Training, Speech & Hearing Disorders 18:273, 1953.

8. Hirsh, I. J.: The Measurement of Hearing, New York, McGraw-Hill Book Company, Inc., 1952.

Case Reports

HYSTERICAL DYSPHAGIA WITHOUT ANEMIA

ADNEY K. SUTPHIN, M.D.
and
PORTER P. VINSON, M.D., Richmond, Va.

HYSTERICAL dysphagia, or the so-called Plummer-Vinson syndrome, is characterized by difficulty in swallowing solid foods, anemia of the hypochromic microcytic type, glossitis, fissures at the corners of the mouth, and, frequently, enlargement of the spleen.

The condition is seen in white women only and especially in those of Scandinavian ancestry. Only three patients with this condition have been observed by us in Virginia in the past 18 years, whereas in Minnesota, where the population consists of a large percentage of persons of Scandinavian descent, the syndrome is much more frequently observed. Many cases have been reported from Scandinavia, with fewer from the British Isles, and almost none from other localities.

The majority of patients are married, more than 40 years of age, and edentulous. Symptoms may have begun suddenly or gradually. Some patients date the onset of symptoms to choking on a pill or some type of solid food. After the initial symptom solid foods, especially meat and green vegetables, are avoided, and gradually anemia and evidences of vitamin deficiency become noticeable. Any solid food that is eaten is chewed until liquefied, and as much as two hours is required to ingest an ordinary meal. Fear of choking on a particle of solid food is the primary cause of the disturbance, and dysphagia of fantastic proportions is a frequent result. One patient could eat ice cream, but not strawberry ice cream, "because of the seeds." Another enjoyed buttermilk but found it necessary to strain out the particles of butterfat to avoid choking.

Enlargement of the spleen occurs in a fairly large percentage of patients suffering from this condition, and the existence of splenic anemia may be suspected. Glossitis is almost always pronounced, and the appearance of the tongue is similar to that of patients having pernicious anemia. Soreness of the mouth and throat and cracks at the corners of the mouth are almost always present.

Roentgenoscopic examination of the esophagus is usually unsatisfactory because of the reluctance of patients to swallow a thick suspension of barium. When the examination is made without difficulty, obstruction is usually not observed, but occasionally a stricture or "web" is suspected. The dramatic relief from symptoms following passage of a sound through the esophagus over a guiding thread and the restoration of the blood picture to normal with recession in the size of the spleen have not been sufficient to convince all those who have observed these patients that the condition is emotional in origin. Some observers believe that anemia from iron deficiency is the primary factor in causing the syndrome and that resulting atrophic changes in the mucous membrane of the esophagus produce stricture. Others have believed that a "web" of congenital origin has been responsible for dysphagia. Relief from difficulty in swallowing is therefore attributed to dilating an organic stricture. We believe that passage of a sound through the esophagus in these cases relieves symptoms by suggestion only and that organic narrowing of the esophageal lumen has not been present in any patient whom we have treated.

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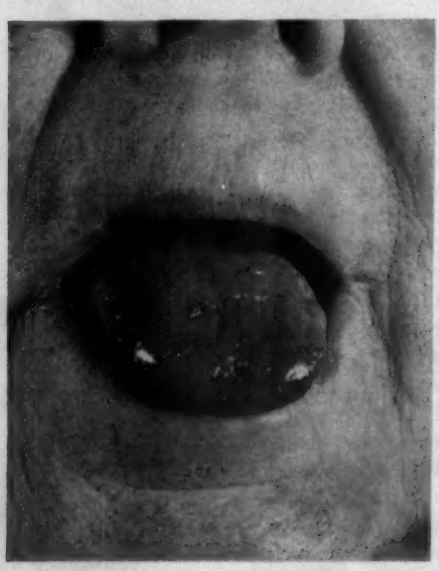
Associate in Medicine (Dr. Sutphin) and Professor of Bronchoscopy, Esophagoscopy and Gastroscopy (Dr. Vinson), Medical College of Virginia.

A recent patient whom we have observed seems to substantiate this conclusion, as she had symptoms and most of the signs of hysterical dysphagia without anemia.

Report of Case

The patient, a widowed white woman, 68 years of age, was first seen on Oct. 12, 1954. Her complaints were soreness of the mouth and throat and inability to swallow solid food. At 2 years of age she had had an attack of acute anterior poliomyelitis with bulbar and spinal involvement. Apparently there was difficulty in swallowing at that time. She eventually completely recovered from the bulbar involvement, although weakness persisted in her right hand for a considerable period of time. She was well thereafter until the age of 38 years, when suddenly on awakening one morning she felt that she had a constricting band around her throat that prevented swallowing. From then until the time of treatment here she had noted continuous difficulty in swallowing any type of solid food, especially meat. She could not remember having eaten beef in any form. Occasionally she would eat a small piece of tender chicken or fish, but this had to be chewed until it was completely disintegrated. She was very fond of green leafy vegetables and continued to eat them although they required mastication for an hour or more before being swallowed. Her weight of 142 lb. had been maintained as the result of ingesting large amounts of hard candy. Her bowel movements were regulated by the use of Agarol (a fluid emulsion of mineral oil and an agar-gel, with phenolphthalein). She had not been able to swallow a pill for many years. Twelve years prior to her examination here, roentgenoscopic study of the esophagus had not revealed any abnormality. Subsequent similar examinations had been reported normal, but a recent study seemed to reveal a narrowing at the pharyngoesophageal juncture. Her mouth and throat had been sore and the tongue smooth and reddened for at least a year. Fissures at the corners of her mouth had been present for many years (Figure). She was edentulous and had worn complete dentures for 15 years. The spleen was not palpable, and the general physical examination was otherwise normal. Complete examination of the blood revealed normal findings. The hemoglobin estimation was 14.9 gm/100 cc., the red blood cells numbered 5,330,000, and the white cells 6500. Differential count of the white cells did not reveal any abnormal features.



On Oct. 19, 1954, an esophageal sound, size 45 F., was passed over a guiding thread into the stomach without detecting obstruction. When the patient was seen the following day, she related that after treatment she had eaten various types of solid food at a normal rate and without evidence of obstruction to swallowing.

A year later the patient was reported to be normal in appearance and had not noted further difficulty in swallowing.

BIBLIOGRAPHY

Vinson, P. P.: Hysterical Dysphagia, Minnesota Med. 5:107-108 (Feb.) 1922.

Clinical Notes, New Instruments and Techniques

TRIAL PRODUCTION OF A NEW SPATULA-LIKE RASPATORIUM FOR LIGATING LARGER BLOOD VESSELS

RYUICHI KUBO, M.D., Fukuoka, Japan

There are many cases in otorhinolaryngological practice in which the ligature of larger blood vessels of the neck becomes necessary. For this purpose Déschamps' needle has been exclusively used, but the original type is too large for the surgery of the neck, and a smaller one has been devised in Japan. In order not to injure the blood vessels (e.g., external carotoid artery or internal jugular vein) to be ligated, they must at first be separated carefully from the surrounding tissues, and then the Déschamps needle is applied.

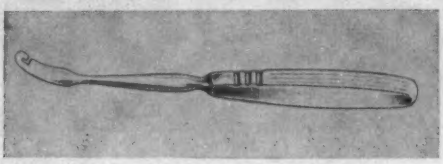


Figure 1

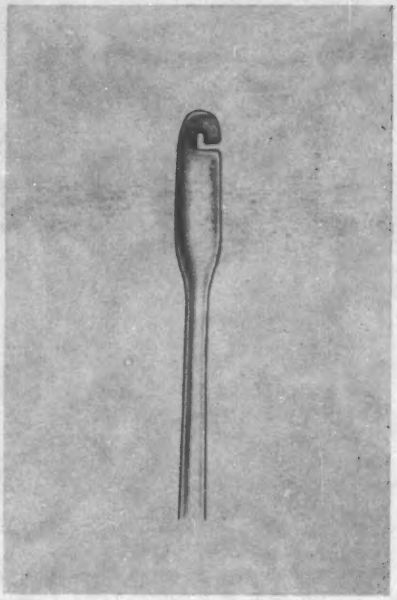


Figure 2

Accepted for publication March 2, 1956.
From the Ear, Nose, and Throat Clinic, Faculty of Medicine, Kyushu University.

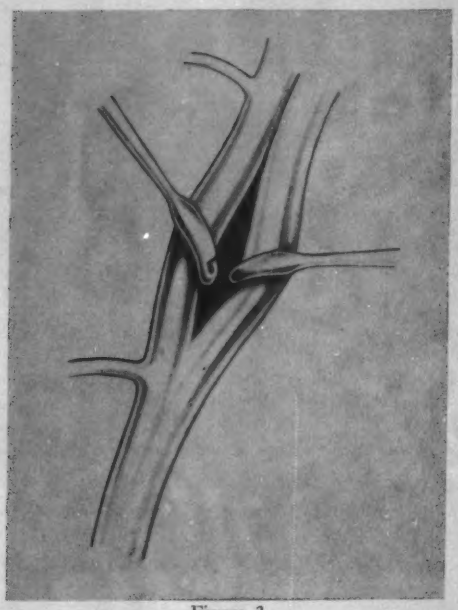


Figure 3

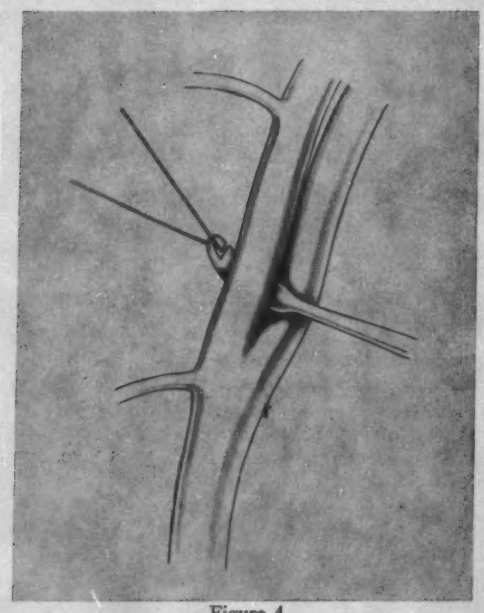


Figure 4

I have devised a new instrument for ligating larger blood vessels more safely and simply. It is a thin spatula-like curved raspatorium with a notch (Figs. 1, 2). As its apex is blunt, the operation of separating the blood vessel is carried out in utmost safety. After separation of the blood vessel with this raspatorium, the ligature thread is easily put into the notch of the raspatorium, whose apex is protruded at the opposite side of the blood vessel (Figs. 3, 4). Thus the ligating procedure is carried out more safely and simply, and it is to be noted that this raspatorium serves for the Déschamps needle.

Abstracts from Current Literature

Ear

THE TECHNIC AND RESULTS OF TYMPANOPLASTY. HORST WULLSTEIN, Ann. oto-laryng. (Paris) 72:764, 1955.

In discussing the chronic suppurations of the middle ear, the author states that he has employed a tympanic flap following his mastoidectomy. His purpose is fivefold: (1) to reconstruct, either partially or totally, the tympanic membrane; (2) to reform an air-bearing tympanic cavity covered by mucosa but without epidermis; (3) to reestablish the continuity of the ossicular chain; (4) to disengage the membrane of the round window in case of its closure, either by cicatrization or by bony overgrowth of the niche, and to be assured that it remains free, and (5) to implant a subsidiary mucosa if necessary. The author then describes his technique, in which he uses not only the flap from the external auditory canal but supplements it by a free graft, either retroauricular or from any other area. Stress is made of the importance of maintaining a freely movable round window. This is important to improve the hearing loss. The author has classified a series of 300 cases in three categories. Cases belonging to Category 1 are those in which he is able to establish (prior to the operation) that the two windows function freely. This can be judged by the aid of an artificial membrane covering the perforation of the tympanic membrane. If there is an appreciable auditory gain, it probably indicates that the entire ossicular chain is both conserved and functioning normally. This type would unquestionably be best benefited by this operation. The third category includes those cases where there is a solid closure, either by connective tissue or by bone, in the niche of the round window. Results in this group of cases are not too favorable. Category 2 includes the intermediate cases. Here there may be some pathology of the mucous membrane of the round window, which can be removed, or there may be an immobilization of the stapes and other pathology, which will yield to the usual technique. The author is extremely enthusiastic about his results, particularly those in Category 1, wherein there has been a marked increase in the hearing acuity. He feels that the operative principles of the tympanoplasty can permit a surgical repair, both anatomically and functionally, in chronic middle-ear infections.

PERSKY, Philadelphia.

OTOLOGIC COMPLICATIONS OF CRANIAL TRAUMA. ROBERT BOURGEOIS, Ann. oto-laryng. (Paris) 72:869, 1955.

The author discusses cranial trauma with its relation to the temporal bone. Fractures of the skull present few symptoms in the external meatus. However, evident pathology due to trauma of the skull can be evidenced by lesions of the tympanic membrane and hemorrhage in the tympanic cavity. These may eventually influence hearing, and it is extremely important to evaluate the presence or absence of a rupture of the tympanic membrane, particularly from the medicolegal standpoint. Secondary infections may also occur. The prognosis is important, and it is influenced by the (*a*) disruption of the ossicular chain, (*b*) cicatricial process, (*c*) adhesions in the middle ear, and (*d*) the presence of secondary infections.

Longitudinal fractures of the temporal bone produce auditory impairment, particularly of the upper tones. Perpendicular fractures usually produce total deafness, with an unresponsive labyrinth to the caloric test. The author also discusses deafness in cranial injuries without fracture. The deafness is usually bilateral, and it usually involves the upper registers. Equilibrium is also affected by cranial injuries. It may be either of a peripheral nature, wherein the symptoms are temporary and easily compensated, or of a central nature, where the symptoms are prolonged and compensation is poor. In perpendicular fractures of the temporal bone or fractures in a paralabyrinthine area, there is unilateral destruction of the labyrinth, which is total and is associated with total deafness. The author also discusses the postconcussional symptoms, which may be due to a temporary edema of the cerebromeninges. 1. The lesions may be benign, where there is a bilateral hyperexcitability of the labyrinth. 2. In other lesions there is a cerebral contusion and the symptoms are more serious and are accompanied by neurologic manifestations, such as hemiplegia, manoplegia, involvement of cranial nerves, etc. Here the vestibular signs can be extremely variable. 3. Radicular lesions are usually unilateral, with the loss of hearing and a perception type of deafness without recruitment and a hypoexcitability of the vestibule.

The above article was reedited and published by Dr. Maurice Bouchet in the memory of his friend Robert Bourgeois.

PERSKY, Philadelphia

MOBILIZATION AT THE FOOTPLATE OF THE FIXED STAPEDIAL FOOTPLATE. SAMUEL ROSEN, Acta oto-laryng. 45:532 (Nov.-Dec.) 1955.

Rosen described in 1952 a technique of mobilization of the stapes which he now refers to as the indirect method. In this technique pressure is exerted posteriorly against the anterior aspect of the neck of the stapes until the fixed footplate is broken loose from its bed. The direct method is one of prying loose the footplate at one, two, or three places at the footplate itself. The exposure technique is the same as that in the earlier operation, except that it is now essential in all cases to remove 2 to 3 mm. of the edge of the posterior bony canal wall. This permits a full view of the incus, long process, incudostapedial junction, etc. At first the indirect method of mobilization is tried. If this fails the direct method is applied. In this way it is possible to achieve successes in the types of situations that were failures by the indirect method.

Rosen presents the indications for the use of the method and 10 illustrative cases.

ROBERT LEWY, Chicago.

IDIOPATHIC HEMOTYMPANUM. EMILIO G., VAQUERO, GARRIDO, AND ANGEL ORTEGO GARCIA, Acta oto-rino-laring. ibero-am. (Barcelona) 6:427, 1955.

The authors present a discussion of hemotympanum, or blue tympanic membrane. They think that perhaps this condition is produced by an obstruction to the Eustachian tube with a marked hyperemia ex-vacuo in the tympanic cavity, which produces a hemorrhagic condition. Paracentesis may have to be repeated with Eustachian inflation. The authors present a case in which, although recurrences did occur, they were finally able to effect a complete cure with repeated paracentesis and inflation.

PERSKY, Philadelphia.

IONTOPHORESIS OF THE INTERNAL EAR AND ADJACENT AREAS WITH INTRATYMPANIC INJECTIONS. RA MARIO RIUS, An. oto-rino-laring. Uruguay 25:49, 1955.

This is a preliminary contribution concerning iontophoresis for hydrops of the labyrinth and the vascular functions of the internal ear. This work is a further modification of similar reports on both the intratympanic action of various solutions and the effect of ionization on the internal ear. The author injects tolazoline hydrochloride (Priscol) supplemented by the hyaluronidase Wydase. Tolazoline acts as a vasodilator, while the Wydase facilitates diffusion of tolazoline into the internal ear, improves the local nutrition, improves any blood stagnation about the capillaries, and influences the endolymph and the hydrops.

His technique originally was to anesthetize the tympanic membrane with a small cotton pledget saturated with Bonain. However, he felt that the phenol present in this solution had a tendency to produce minute perforations in the tympanic membrane and, consequently, replaced this solution with that which was recommended by Trowbridge, namely, 10% cocaine hydrochloride in pure aniline oil. After the anesthetic he injects 0.1 cc. of tolazoline and 0.1 cc. of a fresh solution of hyaluronidase intratympanically. He inserts a specially constructed ear speculum into the external canal and fills the canal with a cotton pledget saturated with 12 drops of tolazoline. The positive pole of the galvanic current is connected to this ear speculum and the negative pole to any other area. The current is turned on to 1 ma. The initial duration is 10 minutes, but this can be increased to 15 minutes and, finaliy, to 30 minutes. Sometimes he may even increase the quantity of tolazoline to 0.2 cc. intratympanically and may also supplement the procedure by an oral administration of tolazoline on the day of the treatment.

The author presents a series of 14 patients with rather interesting results. There were three cases of typical Ménière's disease in which the deafness, tinnitus, and vertigo all improved. There were seven cases of hydrops with or without initial vertigo, five had a definite improvement in hearing acuity, and in six there was marked improvement in tinnitus. There were four cases of Ménière's syndrome without hearing defects; three of the four presented improvement both in the tinnitus and in the vertigo.

The author further discusses the value of this technique in Ménière's disease and evaluates the effect of tolazoline on the various pathologic disturbances that may be responsible for this syndrome. In conclusion, he has shown that in Ménière's disease, in hydrops, and in

Ménière's syndrome without deafness favorable results have been obtained. These results are definitely better audiometrically than those obtained by other therapeutic measures. He has shown that angiospasm is an important factor in Ménière's syndrome, and he feels that it is also possible to localize in the labyrinth all the tests of recruitment, the binoral balance test, the Umbral difference, speech intelligibility, aural diplacusia, fatigue, and many other problems in audiology. Iontophoresis can be used as an important therapeutic test in the differentiation of Ménière's syndrome without hydrops from the vertiginous syndrome of other pathologic origins, such as neurologic and otitic infections, tumors, trauma, allergy, and toxic or degenerative processes. If time will prove the good results obtained by iontophoresis in controlling the vertigo of Ménière's disease, there will be no justification for medical treatment (streptomycin sulfate) or for surgical destruction of the internal ear (alcohol, coagulation, ultrasonic stimulation, and various operations on the membranous canal), which would sacrifice the hearing and yet not guarantee any results to the patient for the other ear. Finally, the author states that iontophoresis of the internal ear is not a therapeutic panacea and that it will always require a careful observation of the patient by the otologist, neurologist, and internist.

PERSKY, Philadelphia.

THE AMINO ACIDS IN THE PERILYMPH FLUID. E. ANTONINI, V. CASORATI, AND S. CRIFO, Rev. laryng. (Bordeaux) 77:59 (Jan.-Feb.) 1956.

The authors have made a study of the various amino acids found in the perilymph fluid in both cats and horses. Basically this was an attempt to determine whether or not perilymph was a derivative of the blood or the cerebrospinal fluid. The results of their work on cats was unsatisfactory. However, in horses they were able to determine the presence of the following amino acids in the perilymph fluid: aspartic acid, glutamic acid, phosphocolamine, glycine, serine, taurine, alanine, glutamine, lysine, arginine, aminobutyric acid, proline, leucine, tyrosine, phenylalanine, and cystine. The study was made by chromatograms, and they found that free amino acids were found in the same quantity in the perilymph as was found in the blood, but in higher concentration than in the cerebrospinal fluid. Furthermore, there were definite qualitative differences; viz., glycine, taurine, proline, and cystine were quite evident in both the perilymph and the blood but were either minimal or absent in the cerebrospinal fluid. The authors believe from these observations of the amino acids that the perilymph is closely related to the blood but differs greatly from the cerebrospinal fluid.

PERSKY, Philadelphia.

ROENTGEN DIAGNOSIS OF CHOLESTEATOMA. GEORGES GUILLEN AND GEORG T. VOGELSANGER, Rev. laryng. (Bordeaux) 77:92 (Jan.-Feb.) 1956.

The authors discuss cholesteatoma and its anatomicopathologic importance. They outline the various roentgen techniques which would help in the proper interpretations of its presence. They recommend Schüller's position, Chausse's position, and the transorbital position. To these may be added Stenver's position and, finally, unilateral tomography. They stress the importance of the otologist personally interpreting the roentgenograms and correlating the findings with the clinical findings.

PERSKY, Philadelphia.

Pharynx

TREATMENT OF PHARYNGEAL DIVERTICULA. M. SPENCER HARRISON, J. Laryng. & Otol. 70:136 (March) 1956.

Treatment of pressure diverticula of the esophagus above the cricopharyngeus muscle by means of endoscopic division of the common wall between the pouch and the esophagus with diathermy is discussed. It is felt that this is the treatment of choice in many such diverticula as compared with the more publicized external approach and excision.

It is pointed out that the external approach is not without serious complications, in spite of antibiotic therapy. These complications are briefly discussed. A discussion of the mechanism of formation of the diverticulum and the structure of the formed pouch is presented. A historical review of the subject gives Mosher credit for first describing division of the septum between the pouch and the esophagus by endoscopic approach in 1917, but Mosher did not utilize the cautery. He had six cases thus treated in a 1929 report, with no complications, but a later patient of Mosher died of mediastinitis. Dohlman used diathermy to divide the wall in

39 patients, with neither complications nor recurrences, in a 1949 report. Other authors with similar experiences are cited.

The method of preparation of the patient and the technique are described. The author reports 15 cases thus treated, with details of 5 representative cases of his group. He feels that there are special advantages of this type of treatment in elderly or otherwise poor-risk patients, and states further that endoscopic division of the party walls by diathermy is the treatment of choice when the sac is large or extending behind the sternum.

LE JEUNE AND STEVENSON, New Orleans.

Larynx

TREATMENT OF EPITHELIOMA OF THE LARYNX AND OF THE PHARYNGOLARYNGEAL SPACES. H. DANCOT AND T. BLAVIER, Acta oto-rhino-laryng. belg. 9:443, 1955.

Malignancy of the larynx and pharyngolaryngeal area has been considered from the standpoint of anatomic location of the lesion, macroscopic appearance, the degree of local extension, and the presence or absence of metastatic adenopathy. 1. Lesions in the larynx involve the cord, the ventricular bands, the laryngeal aspect of the epiglottis, ventricles, and the subglottic region. 2. Pharyngolaryngeal lesions include the aryepiglottic folds, the free portion of the epiglottis, the pharyngoepiglottic folds, the posterior and lateral walls of the pharynx, the vallecula, the arytenoids and the interarytenoid space, and the pyriform sinus. The classification of the lesion as to whether it is capsulated, infiltrative, or ulcerative is an important factor in the prognosis.

In the interpretation of the value of any method of treatment, an evaluation of the prognosis would naturally depend upon the extent of the lesion. Using the classification that was proposed at the International Congress in Copenhagen (1953), the authors classify the various lesions as (1) wherein the lesion is limited to the mucous membrane, (2) where the tumor has infiltrated but has not caused too great a fixation of the larynx, (3) where the tumor has involved the larynx but the palpable glands are freely movable, and, finally, (4) where there has been involvement of the skin, the glands are fixed, and there is a metastasis at a distance from the lesion.

The authors have evaluated the results obtained by the technique of (1) Coutard and of (2) Baclesse. In Coutard's technique the roentgen exposure is increased to a dosage of 4000-5000 r. The duration of treatment is five weeks. This technique is extremely intensive, produces violent reactions (both of the mucosa and of the skin), and is also associated with many general side-reactions. The method of Baclesse does not limit the therapy to five weeks. There is a higher dosage administered to the tumors than in the technique of Coutard: 6000-9000 r. Secondly, there is a maximum protection to the healthy skin. Furthermore, the treatment is extended for a longer duration of time, two months or more, increasing the number of portals of exposure and, finally, narrowing the field of irradiation.

Surgical treatment depends upon the type of lesion. This varies from a simple cordectomy by laryngofissure to the most extensive pharyngolaryngectomy. Where there is a simple localization of the lesion, surgery alone may suffice, particularly in the absence of glandular involvement. On the other hand, a combined therapy of surgery and roentgen rays is sometimes indispensable. In comparing the results, the authors have found that after three years 38% of their cases showed an apparent cure by the Baclesse technique as against the 16% cure in the group treated by the method of Coutard. Side-effects and post-therapy complications were definitely less when the technique of Baclesse, was used. In the group that was treated by a combination of surgery and roentgen therapy the comparison does not hold, since most of the patients were treated by the method of Coutard.

In comparing the results of roentgen therapy alone and that of surgery plus roentgen therapy, they found that where there is an indication for surgery the severity of the case is definitely greatest and the evaluation of relative value is questionable, even though they have figures of 60% cure by surgery in cases of simple laryngofissure or partial laryngectomy. But in cases of total laryngectomy there was a 53% cure in five years, which compares with a 29% cure in cases where roentgen therapy was used alone. In their conclusions they feel that for simple localized lesions in the larynx roentgen therapy of the Baclesse method alone is sufficient. However, where the lesions have extended beyond the larynx proper and there is a greater involvement of the glandular system, the method of choice is a combination of surgery and roentgen therapy.

PERSKY, Philadelphia.

Nose

BRONCHIECTASIS—THE NOSE AND ALLERGY. E. HUIZINGA, Acta oto-rhino-laryng. belg. 9:381, 1955.

The author discusses the subject of bronchiectasis from the pathologic-anatomic standpoint. He feels that atelectasis plays an important role in some of these problems. The development of bronchiectasis is essentially a mechanical problem. There are two general groups: (1) that of a local cause, wherein there is frequently a unilateral condition with a circumscribed bronchiectasis, and (2) that which is more frequently bilateral and diffuse and is often associated with a bronchial stenosis, foreign bodies, tumors, and tuberculosis.

He describes the different types of pathology and symptomatology under these two classifications, and he feels that in diffuse bronchiectasis allergy plays the most important role. More attention should be given to the possibility of pulmonary resection in some of these cases of diffuse bronchiectasis.

PERSKY, Philadelphia.

A REPORT ON THIRTY-FIVE CASES OF MALIGNANT TUMOURS OF NASOPHARYNX. DAVID STEVENS, J. Laryng. & Otol. 70:147 (March) 1956.

This is a comprehensive review of the subject of malignant neoplasms of the nasopharynx, based on a statistical analysis of all of the cases treated at the Cardiff Royal Infirmary from January, 1948, to August, 1954. No new methods of treatment are described. Rather, this is a very thorough description of this class of neoplasms as seen and as treated by the author in the recent past. The article is subdivided into a dozen numbered headings, as follows: (1) Age and Sex Incidence, (2) Histology, (3) Site of Growth and Routes of Extension, (4) Symptoms, (5) Duration of Symptoms, (6) Two Illustrative Cases, (7) Examination of the Nasopharynx, (8) Differential Diagnosis, (9) Aids to Diagnosis, (10) Associated Lesions, (11) Treatment, and (12) Results of Treatment. Associated with these headings nine illustrative tables are presented which aid the statistical presentation of the 35 cases. Under the heading Examination of the Nasopharynx, the author describes a method of examination and a corresponding physical sign, which he feels is useful and which, he states, is unrecorded heretofore in the literature. The method of examination is to occlude each side of the nose individually and listen to the sound made by the breath passing through the unoccluded side. If there is any suggestion of infiltration of the walls of the nasopharynx due to a growth, there will be a unilateral "cavernous nasal breathing."

This paper is a well-presented general discussion of a lesion that is often missed or diagnosed late, and it is recommended reading for physicians in all branches of clinical medicine.

LE JEUNE AND STEVENSON, New Orleans.

Miscellaneous

HYPOADRENOCORTICISM IN OTOLARYNGOLOGIC SURGICAL PROCEDURES. JOHN W. TINTERA AND HERBERT B. GOLDMAN, N. Y. J. Med. 56:872 (March 15) 1956.

Tintera is with the Endocrine Clinic, St. John's Riverside Hospital, Yonkers, N. Y., and Goldman, the Otolaryngology Division of Kings County Hospital, Brooklyn. Their thesis aims to prove that many children (and some adults) are constitutionally inferior and do not well withstand the stress of surgery, especially tonsillectomy. There is inadequate corticoadrenal response to mental and physical trauma because of inheritance. If either parent has hypoadrenocorticism, the child is likely to inherit this condition. Careful personal and familial histories are highly important in evaluating glandular integrity. "Our professional status should be more than that of a surgical technician." If hypoadrenocorticism is present, the authors say resistance to viral infections, for example, is lowered. "It is only the debilitated child who succumbs to coccal infections." H. Selye is quoted as saying that disease consists of two elements, damage and defense. "Intravenous hydrocortisone is the agent of choice in surgical procedures because approximately 80 per cent of the corticosteroids found in adrenal venous blood is hydrocortisone." In later sentences: "If as little as 50 mg. of hydrocortisone are given, adrenal cortical suppression is imminent. Changes in the adrenal may persist for six months or permanently after hydrocortisone therapy has been terminated."

How does one determine hypoadrenocorticism? One tries to elicit excessive fatigue or weakness, nervousness, irritability, mental depression (cyclic or otherwise), and, most important, fears or apprehensions, frustrations, and tendencies to negativism. In children we

are usually dealing with thin, short-statured persons, very fearful and sickly. They are the mouth breathers, with the typical adenoid facies, receding chin, high palatal arch, cervical lymphadenopathy, allergies, and long, narrow chest with acute costal angle. "Since we consider all allergic manifestations to be on an adrenocortical deficiency basis, and with the history of craving for salt and carbohydrates and the typical sex characteristics, diagnosis could be facilitated if we had one test for corroboration." The authors say we have only to show hypoglycemia. Here we have a sudden fall in blood, sugar, dilatation of the pupils, circumoral pallor, fine tremors of the extremities, involuntary twitchings, and tachycardia. "Very often, before surgery is attempted, it is necessary . . . to give injections of from 2 to 5 cc. of adrenal cortical extract at weekly intervals for . . . several months plus a low-carbohydrate diet."

As for the thymus: "A persistence . . . beyond a certain age inhibits the normal development of the adrenal cortex, and, conversely, it is assumed that the administration of adrenal cortical extract causes a regression of the thymus gland. We are in favor of this procedure over x-ray radiation since the latter only obliterates the thymus without treating the hypoadrenocorticism. . . . During prolonged operations . . . for malignancy of head and neck, adrenal cortical extract may be added to the intravenous infusions."

Oozing after tonsillectomy is said to be due to capillary fragility from endocrine disturbances, and it may be diminished by adequate hormonal therapy. The present study comprises some 552 tonsillectomies in children, of whom 120 received Eschatin. The authors have estimated that roughly one person in six has an adrenocortical deficiency of varying degree, not all of whom necessarily require treatment. "Every patient coming to operation must now be questioned as to any medication they have taken in the recent past. . . . After an intravenous injection of adrenal cortical extract (5 to 30 cc.), there was an abrupt change in condition within a few minutes." Nausea was gone, cold perspiration stopped, the skin became dry and normal in color, and a renewed feeling of self-confidence supervened.

To summarize, one must diagnose a hypoadrenocortic patient by taking complete personal and family histories associated with a complete physical examination. These persons respond to operations poorly, and even a minor procedure may be fatal. But when whole adrenal cortical extract is administered, they are able to withstand the alarm reaction and stage of resistance incident to surgical stress. Cortisone and hydrocortisone are contraindicated because of the many dangerous side-effects, the most important of which is suppression of adrenal cortical function. Severe reactions following cortisone and hydrocortisone therapy are now being reported in the surgical literature. But aqueous adrenal cortical extract is not dangerous and is physiologic. It serves to reduce poor surgical risks to fairly uncomplicated operative and postoperative normal sequences.

ABSTRACTER'S COMMENT: Many physicians of experience have a dread of tampering with the endocrine system, since it is so mutually interdependent of one gland on another, and of one on the whole chain of these glands. From this essay one might fairly conclude that almost every patient about to undergo surgery, especially ENT surgery, should be studied by an endocrinologist and be injected with the super kidney glandular extract. It seems that some 80% would fit into such a pattern, but here, as elsewhere, statistics may be more imaginary than real and not dependable, certainly not in matters of life and death.

I. W. VOORHEES, New York.

News and Comment

PERSONAL

Dr. Eugen Grabscheid Receives an Award.—On May 21, 1956, at The Harlem Eye and Ear Hospital, Dr. Eugen Grabscheid received the Doctor Charles Bramman Meding Award. This Award, granted annually in memory of Dr. Meding, was bestowed upon Dr. Grabscheid in recognition of his outstanding clinical service at The Harlem Eye and Ear Hospital and for his presentation of programs of study in the interest of the advancement of the art of otolaryngology.

Dr. Dean M. Lierle Receives an Award.—The Newcomb Award of the American Laryngological Association was presented to Dr. Dean M. Lierle, of Iowa City, for his significant contributions to laryngology and rhinology and to the American Board of Otolaryngology.

ANNOUNCEMENTS

Laryngology and Bronchoesophagology Course.—The next Laryngology and Bronchoesophagology Course to be given by the University of Illinois College of Medicine is scheduled for the period Nov. 5 through Nov. 17, 1956. The Course is under the direction of Dr. Paul H. Holinger.

Interested registrants will please write directly to the Department of Otolaryngology, University of Illinois, College of Medicine, 1853 W. Polk St., Chicago 12, Ill.

Annual Assembly in Otolaryngology.—The Department of Otolaryngology, University of Illinois College of Medicine, announces its Annual Assembly in Otolaryngology from Oct. 1 through 7, 1956. The Assembly will consist of an intensive series of lectures and panels concerning advancements in otolaryngology; evening sessions will be devoted to surgical anatomy of the head and neck and histopathology of the ear, nose, and throat.

Interested physicians should write directly to the Department of Otolaryngology, 1853 W. Polk St., Chicago 12, Ill.

At the Sixth International Congress of Otolaryngology, which will be held in Washington, D. C., May 5-10, 1957, three Plenary Sessions will be devoted to previously selected subjects. These Sessions are scheduled for Monday, Wednesday, and Friday mornings, May 6, 8, and 10, and the following internationally recognized authorities will open the discussions:

Chronic Suppuration of the Temporal Bone
- Marcus Diamant, Sweden: Anatomy and Etiology
- Luzius Rüedi, Switzerland: Genesis of Cholesteatoma
- Horst Wullstein, Germany: Tympanoplasty

Collagen Disorders of the Respiratory Tract
- Hans Selye, Canada: Introduction
- Michele Arslan, Italy: Upper Respiratory Tract
- Leslie Gay, U. S. A.: Lower Respiratory Tract

Papilloma of the Larynx
- Jo Ono, Japan: Etiology
- Plinio de Mattos Barretto, Brazil: Diagnosis
- F. C. W. Capps, England: Therapy

Pan-Pacific Surgical Association to Hold Seventh Congress.—The Seventh Congress of the Pan-Pacific Surgical Association will be held in Honolulu, T. H., Nov. 14-22, 1957. All members of the profession are cordially invited to attend and are urged to make arrangements as soon as possible if they wish to be assured of adequate facilities.

An outstanding scientific program by leading surgeons, with sessions in all divisions of surgery and related fields, promises to be of interest to all doctors.

Further information and brochures may be obtained by writing to Dr. F. J. Pinkerton, Director General of the Pan-Pacific Surgical Association, Room 230, Young Building, Honolulu, T. H.

Examination to Be Given by the American Board of Otolaryngology.—The American Board of Otolaryngology will conduct its next examination on Oct. 8-11, 1956, at the Palmer House, in Chicago.

GENERAL NEWS

Specialists Certified by the American Board of Otolaryngology.—The following physicians were certified as specialists by the American Board of Otolaryngology at the recent examination held May 6-9, 1956.

Arsuaga, Lorenzo, Puerto Rico.
Ballenberger, Louis P., Chelsea 50, Mass.
Brandow, Edward C., Jr., Albany, N. Y.
Brunelle, Paul, Montreal, Canada.
Burkey, Jordan W., Jr., Springfield, Mo.
Chandler, James R., Jr., Fort Bragg, N. C.
Clopton, James W., Fullerton, Calif.
Dean, John L., Jr., Crockett, Texas.
Devine, Kenneth D., Rochester, Minn.
Ejercito, Napoleon C., Buffalo 15.
England, Myron C., Woodward, Okla.
Gardner, Carl A., Ann Arbor, Mich.
Giotta, Peter J., New York 62.
Goodman, Merrill, New Hyde Park, N. Y.
Grandon, Eugene L., Iowa City.
Hakim, Arcadius H., Boston 14.
Insley, Marion C., Jr., Danville, Pa.
Kyler, Stephen L., Pittsburgh 40.
Langgaard, Charles E., Chatham, N. J.
MacMillan, Bruce B., Pittsburgh 13.
Melucci, Nicola M., Brooklyn 38.
Mettler, Donald C., Ann Arbor, Mich.
Meyers, Edward A., Yonkers 5, N. Y.
Montgomery, William W., Boston.
Obregon, Gonzalo, Iowa City.
Perone, Piero M., New York 21.
Rounthwaite, F. John, London, Ont. Canada
Singh, Harbans, New York 21.
Sinha, Sharda P., Montreal, Canada.
Stephens, William Carey, Tyler, Texas.
Tippins, H. Keener, Fort Walton Beach, Fla.
Walker, Ethan A., Jr., Oklahoma City 3.
Williams, John R., New Rochelle, N. Y.
Wilson, Heston L., Seattle 1.
Zanka, Jaroslav A., Butler, Pa.

Directory of Otolaryngological Societies *

FOREIGN AND INTERNATIONAL

Canadian Otolaryngological Society

(Société Canadienne d'Otolaryngologie)

President: Dr. D. S. Gorrell, 110 Medical Arts Building, Calgary, Alta.
Secretary: Dr. G. Arnold Henry, 328 Medical Arts Building, Toronto.

International Bronchoesophagological Society

President: Dr. Antonio da Costa Quinta, Lisbon, Portugal.
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40.
Meeting: Fourth International Congress of Bronchoesophagology.
President of Congress: Dr. Antonio Carrascosa.

International Congress of Audiology

Place: Leyden, Netherlands.
General Secretary: Dr. H. C. Huizing, 182 Oostersingel, Groningen, Netherlands.

International Course in Paedo-Audiology

Place: Groningen, Netherlands.
General Secretary: Dr. H. C. Huizing, 182 Oostersingel, Groningen, Netherlands.

Pan-American Association of Oto-Rhino-Laryngology and Broncho-Esophagology

President: Dr. Juan Manuel Tato, Azcuenaga 235, Buenos Aires, Argentina.
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40.

Sixth International Congress of Otolaryngology

President: Dr. Arthur W. Proetz, 3720 Washington Blvd., St. Louis.
General Secretary: Dr. Paul H. Holinger, 700 N. Michigan Ave., Chicago 11.
Place: Hotel Statler, Washington, D. C. Time: May 5-10, 1957.

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. John R. Lindsay, Chicago.
Secretary: Dr. Hugh A. Kuhn, 112 Rimbach St., Hammond, Ind.

American Academy of Ophthalmology and Otolaryngology

President: Dr. Algernon B. Reese, 73 E. 71st St., New York 21.
Executive Secretary-Treasurer: Dr. William L. Benedict, 100-1st Ave., Bldg., Rochester, Minn.

American Board of Otolaryngology

President: Dr. Frederick T. Hill, Professional Building, Waterville, Maine.
Secretary-Treasurer: Dr. Dean M. Lierle, University Hospitals, Iowa City.

American Broncho-Esophagological Association

President: Dr. Daniel S. Cunning, 115 E. 65th St., New York 21.
Secretary: Dr. F. Johnson Putney, 1719 Rittenhouse Sq., Philadelphia 3.

American Laryngological Association

President: Dr. LeRoy A. Schall, 243 Charles St., Boston 14.
Secretary: Dr. Harry P. Schenck, 326 S. 19th St., Philadelphia 3.
Place: Washington, D. C. Time: May 3, 1957.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. Dean M. Lierle, Iowa City.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester 7, N. Y.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

SECTIONS:

Eastern.—Chairman: Dr. John A. Murtagh, Hanover, N. H.
Southern.—Chairman: Dr. J. M. Robison, Houston, Texas.
Middle.—Chairman: Dr. William H. Craddock, Cincinnati.
Western.—Chairman: Dr. David D. DeWeese, Portland, Ore.

AMERICAN OTOLOGICAL SOCIETY, INC.

President: Dr. William J. McNally, 1509 Sherbrooke St. West, Montreal 25, Canada.
Secretary-Treasurer: Dr. Lawrence R. Boies, 90 S. Ninth St., Minneapolis 2.

AMERICAN OTORHINOLOGIC SOCIETY FOR THE ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY, INC.

President: Dr. Joseph Gilbert.
Secretary: Dr. Louis Joel Feit, 66 Park Ave., New York 16.

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY

President: Dr. Dean McAllister Lierle, University Hospital, Iowa City.
Secretary-Treasurer: Dr. Michael H. Barone, 468 Delaware Ave., Buffalo 2.
Place: Palmer House, Chicago.

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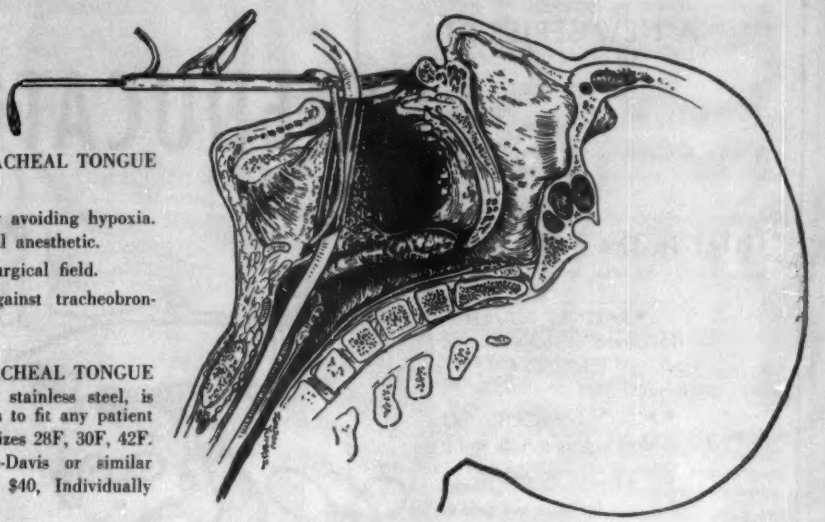

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*Barton, R. T.: Instrument for endotracheal anesthesia during oro-pharyngeal surgery. J. A. M. A. 153:1017 (Nov. 14) 1953.

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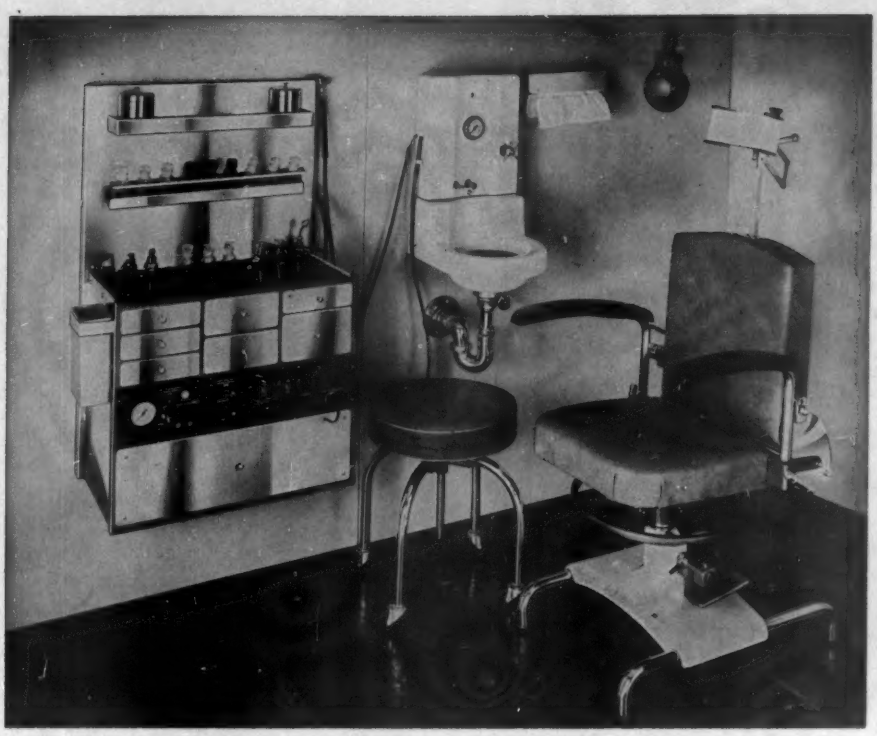

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Chair: Can be placed in horizontal position, will not tip in this position; raised and lowered by large hand wheel; paper covered headrest removable; locked with foot-pedal; bakelite armrests removable. Back locks rigidly in any position. As shown in brown or green. $150.00.

Turning Chair: By adding a footrest and the necessary handles this chair can be used for turning tests. All the additional equipment is easily and quickly removable. Add. $30.00.

Combined Suction and Cuspidor: This is an entirely new piece of equipment. It was designed to eliminate the cost of installing a separate suction. The apparatus is 12" wide and projects 13¼" from the wall. The flushing rim and the suction is furnished with a vacuum breaker and should meet all plumbing requirements. Stop and trap not furnished. Available in ivory, green and white. Price $95.00.

Ceiling Light: A telescopic light attached to the ceiling. May also be attached to base board in back of chair. Available in 4, 5, 6 and 7 ft. (extended). 4 ft. $16.50 (add one dollar per ft.).

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Stool: A soft upholstered stool with easy running ball bearing stem casters. $20.00. Stool with back, $26.00.

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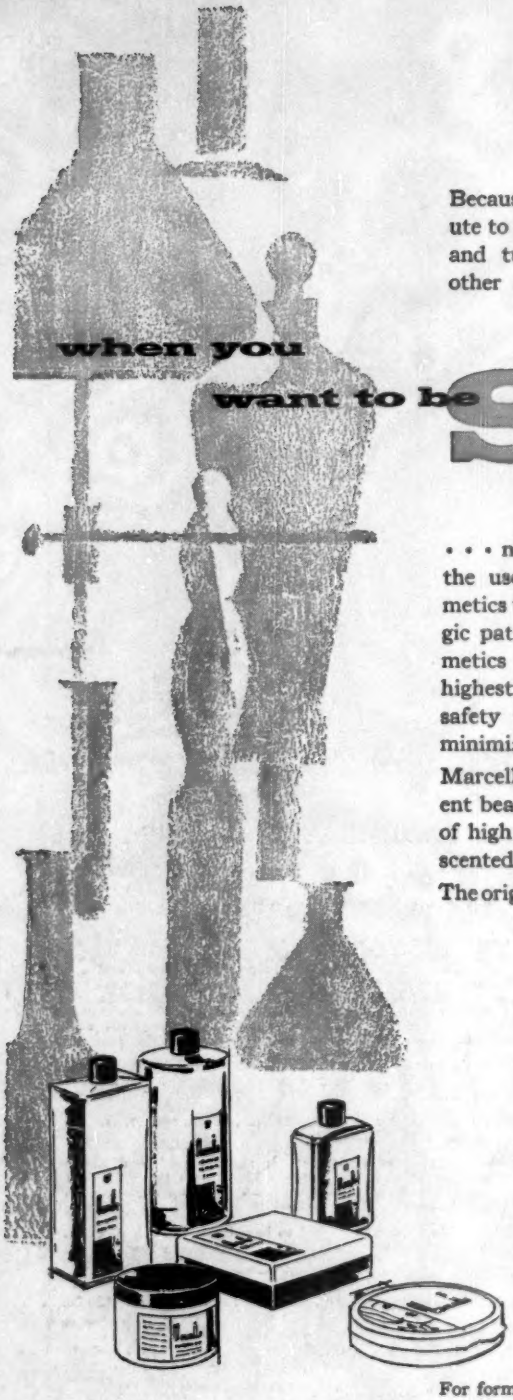

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